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AUGUST 1949

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $10.00

Entered as Second Class Matter Jan. 7, 1919, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

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VOLUME 62 AUGUST 1949 NUMBER 2

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EFFECTS OF ULTRAVIOLET RADIATION ON THE EXPOSED BRAIN

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AND
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WASHINGTON, D. C.

GERMICIDAL radiation of the operating field with ultraviolet rays has contributed much to the asepsis of surgery. Its effects on various tissues have been studied.[1] Hart and Sanger [1a] alone reported the effect of ultraviolet radiation on the exposed brain and spinal cord of 2 dogs. They stated that after exposures to wavelengths of approximately 2,537 angstrom units for one hour no injurious effects were found pathologically.

The purpose of this paper is to report the pathologic changes resulting from five hour exposures in 10 brains of cats having bilateral maximal craniotomies. One hemisphere was shielded from the ultraviolet rays for control purposes, while the other hemisphere was irradiated. The exposures were made in the regular operating theaters of the Montreal Neurological Institute. The brain of each cat was studied pathologically between the third and the fifth postoperative day.

THEORETIC CONSIDERATIONS

For all practical purposes, we are concerned with the effect of ultraviolet radiation in the neighborhood of 2,537 angstrom units, which is the zone of greatest germicidal effectiveness.[2]

It is now generally believed that ultraviolet radiation of wavelengths shorter than about 3,200 angstrom units affects cells by direct action on cellular constituents which absorb these specific wavelengths. The effects are usually considered lethal or injurious, but under special

From the Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute.

Presented at the annual meeting of the American Academy of Neurological Surgery, Hot Springs, Va., Sept. 11, 1946.

1. (*a*) Hart, D., and Sanger, P. W.: Effect on Wound Healing of Bactericidal Ultraviolet Radiation from Special Unit, Arch. Surg. **38**:797-805 (May) 1939. (*b*) Kraissl, C. J.; Cimiotti, J. G., and Meleney, F. D.: Ann. Surg. **3**:161-185, 1940.

2. Coblentz, W. W.: Physical Aspects of Ultraviolet Therapy, J. A. M. A. **111**:419-423 (July 30) 1938.

conditions ultraviolet radiations may induce mutations in somatic cells,[3] fungi,[4] liverwort spores [5] and maize pollen.[6]

It has been shown that both proteins and nucleic acids absorb wavelengths shorter than 3,200 angstrom units, the maximal protein absorption occurring in the neighborhood of 2,800 angstrom units and the greatest absorption of nucleic acid lying closer to 2,600 units.[7] Giese and associates, working with fine and narrow alterations of the radiation spectrum, stated the belief that protein is the principal light absorber and consequently that the effect is localized principally in the cytoplasm.[8]

Maximal epidermal erythema occurs at 3,000 angstrom units.[2] Blum [9] reported that the erythema of sunburn is due to a histamine-like dilator substance released by injured epidermal cells which diffuses to the underlying minute vessels.

In addition, it is interesting to note that the incidence of tumors of the limbus of the eye in the southern states parallels that of human cutaneous cancer.[10] Wavelengths shorter than 3,200 angstrom units produce malignant growths in the cornea of mice.[11]

TECHNIC OF STUDY

Anesthesia and Preparation.—Each animal was anesthetized with intraperitoneal injection of pentobarbital sodium (0.5 mg. per kilogram of body weight). The entire head was shaved, cleaned with green soap, scrubbed with alcohol and prepared with nitromersal (metaphen®).

The mouth was placed astride a small iron bar and the skull held in place with four projecting prongs, one at each quadrant of the skull.

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3. Makenzie, K., and Muller, H. J.: Proc. Roy. Soc., London, s.B **129**:491-517, 1940.

4. Hollaender, A., and Emmons, C. W.: (*a*) Cold Spring Harbour Symposia Quant. Biol. **9**:170-186, 1941; (*b*) J. Cell Comp. Physiol. **13**:391-402, 1939.

5. Knapp, A. E.; Reuss, A.; Risse, O., and Schreiber, H.: Naturwissenschaft. **27**:304, 1939.

6. Stadler, L. J., and Uber, F. M.: Genetics **27**:84-118, 1942.

7. (*a*) Blum, H. F.: Ann. Rev. Physiol. **5**:1-16, 1943. (*b*) Abramson, H. A.: Proc. Soc. Exper. Biol. **43**:410-412, 1940. (*c*) Giese, A. C.: Biol. Bull. **75**:238-247, 1938. (*d*) Hollaender and Emmons.[4a] (*e*) Hollaender, A.; Jones, M. R., and Jacobs, L. J.: Parasitology **26**:421-432, 1940. (*f*) Lander, E. W.: J. Cell. Comp. Physiol. **14**:217-225, 1939.

8. Giese [7e]; J. Cell. Comp. Physiol. **13**:139-150, 1939; ibid. **14**:371-382, 1939; Physiol. Zool. **12**:319-327, 1939; J. Cell. Comp. Physiol. **17**:203-220, 1941; ibid. **18**:1-3, 1941; ibid. **20**:35-46, 1942. Giese, A. C., and Reed, E. A.: ibid. **15**:395-408, 1940.

9. Blum, H. F.: American Chemical Society, Monograph 85, New York, Rheinhold Publishing Corporation, 1941.

10. Ash, J. E., and Wilder, M. C.: Tr. Am. Acad. Ophth. May-June, 3-10, 1942.

11. Grady, H. G.; Blum, H. F., and Kirby-Smith, J. S.: J. Nat. Cancer Inst., to be published.

Operation.—Under sterile conditions, the scalp was reflected in four leaves after a crossed incision was made. The vault of the skull was extensively removed on each side, only a narrow ridge of bone above the medial longitudinal sinus being spared. The dura was opened over the temporal lobe and reflected, exposing practically the whole of the superior surface of the brain on each side, which included at least half the lateral and posterolateral gyri; the anterior, medial and posterior suprasylvian gyri; the medial, anterior and posterior ectosylvian gyri; the coronal gyrus, and the posterior sigmoid gyrus (figs. 1 and 2).

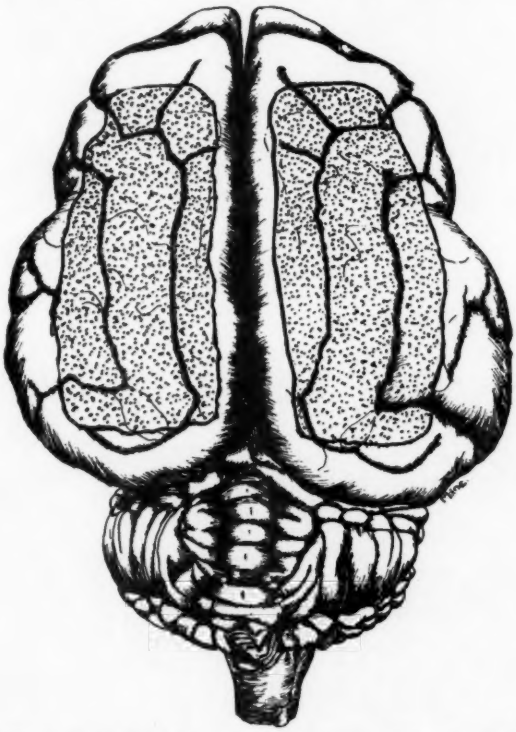

Fig. 1.—Diagram showing the approximate exposure of the cat brain in each experiment. The area of exposure is indicated by the shaded zone. One hemisphere was irradiated with ultraviolet light and the other was shielded in each experiment.

One cerebral hemisphere was exposed to direct ultraviolet radiation of 2,537 angstrom units for five hours, while the opposite hemisphere was shielded by a watch glass and, on one occasion, by lead foil and red paper. On one occasion the irradiated side was covered with a quartz window. The covering material was always elevated 1 to 2 cm. above the surface of the brain to prevent moisture from clouding vision and to allow for gentle irrigation with Ringer's solution in the surrounding gutter between bone and brain, the field being kept uniformly moist. At the end of the experiment the dura was folded back in place over the medial half of the exposure, and the scalp and muscle flap were closed.

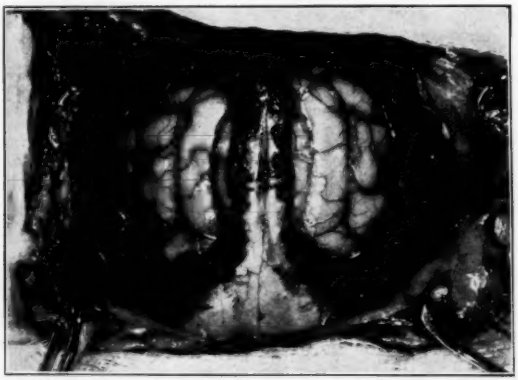

Fig. 2.—The appearance of the brain during exposure. Note the amount of each hemisphere exposed. Dilatation of the smaller arteries and engorgement of the veins are shown on the right.

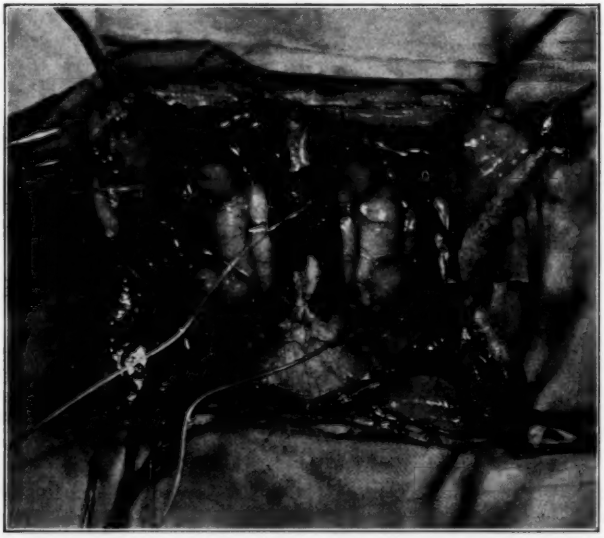

Fig. 3.—The four positions for electroencephalography during the exposure are shown. Cotton pledgets moistened with isotonic sodium chloride solution were used for direct contact.

For 4 of 10 cats electroencephalograms were made before and during irradiation (fig. 3).

Conditions in Operating Room.—The source of untraviolet radiation was the 30 watt (glass 974) General Electric low pressure mercury vapor germicidal lamp in a tube 36 inches (91.4 cm.) long and 1 inch (2.5 cm.) in diameter. According to specifications, this source emits 11,000 milliwatts of lethal radiant energy of which about 80 per cent is in the region of 2,537 angstrom units. The lamp is provided with a suitably formed aluminum reflector, which emits 60 to 80 per cent of the flux emitted by the source when its output is otherwise unrestricted. This lamp emits between 120 and 145 microwatts per square centimeter at 1 meter distance. Two lamps were used, each placed 10½ feet (315 cm.) from the floor and 7 feet (210

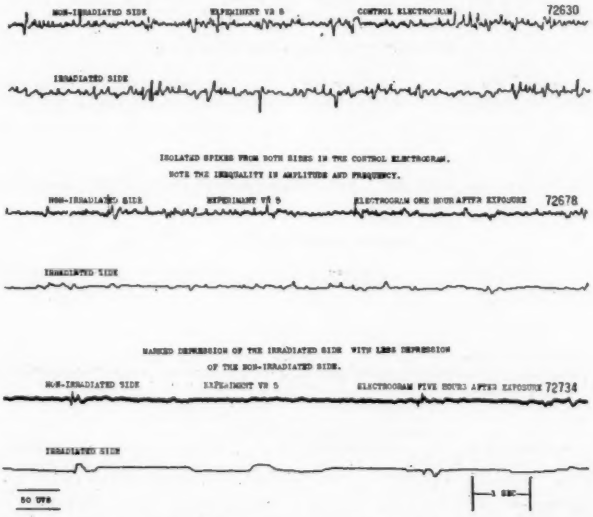

Fig. 4 (cat 5).—Pentobarbital sodium anesthesia. Electrocortograms show an initial depression on the irradiated side within one hour after exposure. The depression became so severe that by the end of five hours the brain waves were absent. Note that the final depression of activity has affected both sides, except for odd potentials, which may represent an artefact.

cm.) from the exposed brain. The center of the lamp had its midpoint over the center of the area of brain to be irradiated, each lamp being parallel to the longitudinal axis of the animal. Visual operating lights were turned off during the experiment, eliminating as much as practicable the factor of heat and ordinary light. When electrograms were made, the protecting cover was removed from the control side in some cases. The ultraviolet generators were then stopped for a brief interval. Surfaces of the brain were kept moist by gentle irrigations with Ringer's solution every fifteen to thirty minutes without removal of filters.

In addition, ultraviolet germicidal wall lamps were used during the exposures. They did not emit any direct rays onto the brain.

RESULT OF OPERATIVE EXPOSURE

Observations During Irradiation and Exposure.—Vascular Changes: The first change noted on gross inspection of the surface of the brain during the exposure on the irradiated side was a slight dilatation of the smaller arterial vessels of the pia, which reached and remained

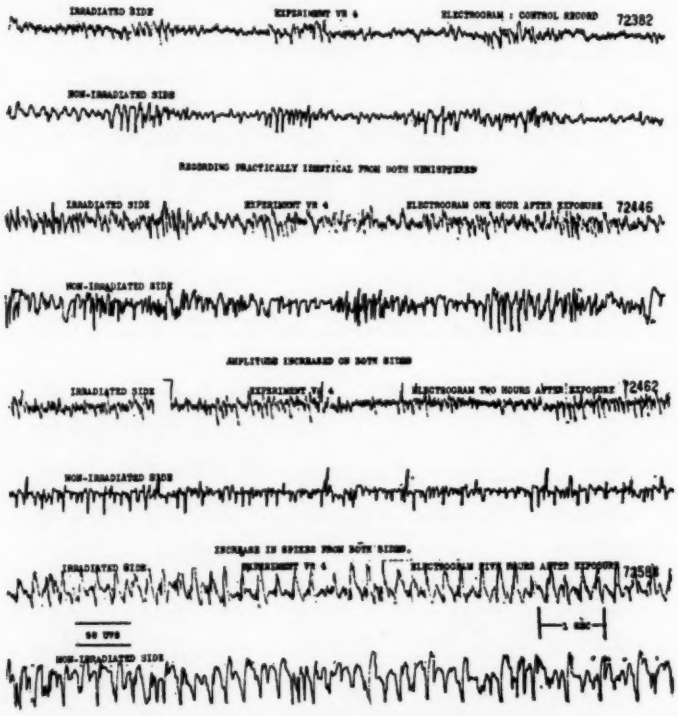

Fig. 5 (cat 4).—Pentobarbital sodium anesthesia. Electrocortograms show the appearance of an electrical seizure after five hours' ultraviolet irradiation. Note that the multiple spikes and sharp waves of large amplitude are conducted to both hemispheres. Slow waves from both sides followed termination of the electrical seizure.

at a certain maximum characteristic for the individual case. It was sometimes apparent that the occasional large artery present in the field had become more prominent and that the ground of the cortical surface had become diffusely hyperemic. Some degree of venous engorgement was sometimes recorded (fig. 2).

The duration of irradiation before the appearance of vascular dilatation was not the same in all animals. It varied from case to case, as did the time required for production of maximal hyperemia. Both of these time intervals were determined merely by direct observation, and the determination of the maximal effect was, of necessity, very rough.

The earliest development of engorgement of pial arterial vessels was noted in case 3 after an irradiation of eighteen minutes, the effect rising to a constant maximum of thirty minutes. The slowest development was in case 4 after two hours and twelve minutes, the effect rising to maximal intensity in three hours and two minutes. In 2 other cases the earliest degree of visible vascular dilatation appeared after one and a half hours and one hour, in 5 cases a questionably definite effect occurred after two and three-fourths hours, fifty minutes, thirty-three minutes, thirty minutes and thirty minutes, respectively. These observations were difficult to make with accuracy, though the time required for the development of the first appearance of hyperemia was fairly accurate.

On the control side, the vessels were questionably affected in case 3 at one hour, with some advance after five hours. In case 4 the larger vessels appeared injected at three hours and two minutes. No vascular changes were noted on the control side in the other cases. This is in disagreement with the observations of Prados and associates,[12] who observed gross circulatory changes, which reached a maximum in about two hours on simple exposure of the brain of the cat to air alone.

In 2 instances the ultraviolet light was turned off for twenty minutes, during which time the vessels over the previously irradiated hemisphere returned to normal. On reexposure to the ultraviolet radiation they quickly resumed their fully dilated appearance.

In cat 2 thirteen cortical hemorrhages were visible at the end of exposure over the irradiated area.

Cerebral Edema: No swelling of the brain during the exposures was noted in cat 1. In cat 2 no swelling was noted on the irradiated side. The control hemisphere was thought to show edema after three hours and thirty minutes, but with proper illumination this impression was not confirmed. In cat 3 swelling was suspected on the irradiated side at forty-two minutes, one hour and five hours, but the brain was considered normal on other examinations. The convolutions on the control side were thought to be more convex, though of similar width, at one hour and thirty-five minutes, and again at five hours.

12. Prados, M.; Strowger, B., and Feindel, W. H.: Arch. Neurol. & Psychiat. **54**:163-174 (Sept.) 1945.

Cat 4 showed possible edema on the irradiated side at two hours and twenty-nine minutes but exhibited no further increase. However, this hemisphere appeared less prominent than the control throughout the experiment. The control side appeared to be possibly more prominent or swollen than the left at fifteen minutes and remained thus until the end of the experiment. Whether this appearance really represented edema on the control side or was merely a question of unequal gross structure cannot be stated with certainty. In any event, it emphasizes the fact that there was no definite swelling on the irradiated side.

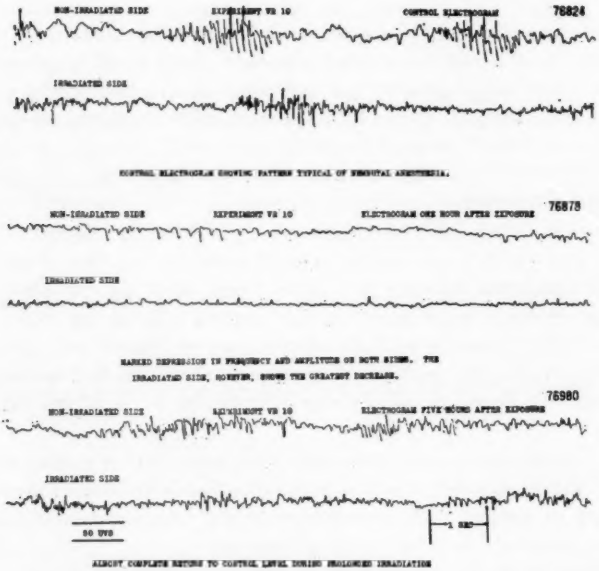

Fig. 6 (cat 10).—Pentobarbital sodium anesthesia. Electrocortograms show an initial depression in frequency and amplification on both sides after one hour, with the greatest depression on the irradiated side. Compare with figures 4 and 5. At the end of five hours the electrogram had returned to the control level.

In cat 5 no edema was recorded. In cats 6 and 8 swelling and slight swelling were noted at two hours and five minutes and one hour and twenty-five minutes, respectively. In cats 7, 9 and 10 swelling was not definitely observed during the exposure.

It is apparent from the foregoing that gross edema on the irradiated side was suspected in only a few instances during exposure. No definite swelling of the control hemispheres was noted in any experiment. It may be added, however, that in all 10 cases edema was present post mortem, being greater on the exposed side.

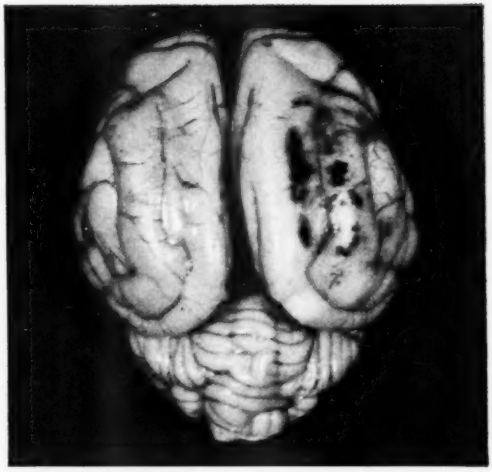

Fig. 7.—Formaldehyde-fixed brain of a cat three days after a five hour period of ultraviolet irradiation of the right hemisphere. Note differences in swelling, as well as the relative absence of swelling over the nonirradiated side. The hemorrhages on the irradiated side were not all visible during exposure.

Clinical Observations: At the end of five hours of irradiation, cat 4 showed electrographic and clinical evidence of a generalized seizure. The animal breathed deeply, the trunk quivered and there were isolated clonic jerks of the legs. Recovery was uneventful. All other animals were asymptomatic during the exposure.

Electroencephalographic Observations: Cortical electrograms were taken during exposure on 4 animals, with electrodes placed directly

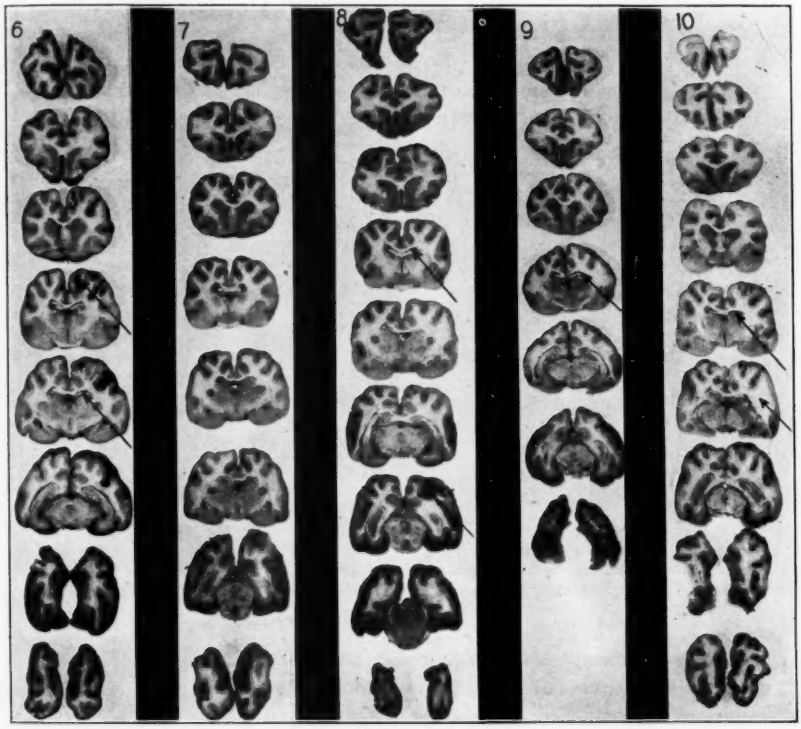

Fig. 8.—Appearance of the cerebral sections prepared three to five days after five hour exposures to ultraviolet radiations. In this series the right hemisphere was irradiated and the left hemisphere shielded from the radiations by glass. Note the general hemispheral edema, the ventricular compression on the edematous side and the presence of a few of the petechial hemorrhages. These sections were made to include, and not to transsect, the petechial hemorrhages, so that they might be included within the microscopic preparations. Note the large hemorrhagic cyst in the white matter of the gyrus adjacent to the midline in *6*.

on the pial surface of the irradiated and the nonirradiated hemisphere (fig. 3). The characteristic changes were as follows: (*a*) cat 5: development of a marked depression in the electrographic activity

after one hour with some slow waves of low amplitude (fig. 4), most marked on the irradiated side, where they persisted until the termination of the exposure: (*b*) cat 4: epileptiform spikes after one hour, followed by a clinical seizure at the end of five hours (fig. 5), and (*c*) cats 9 and 10: depression after one hour and return of brain waves during the exposure on both the irradiated and the nonirradiated side (fig. 6). In the case of cat 9 some variability in the amount of depression is evident.

POSTOPERATIVE OBSERVATIONS

There was no mortality. Some cats showed drowsiness and hypomobility on the first and second postoperative days. One, cat 6, did not eat on the first postoperative day. A tube feeding was attempted,

Fig. 9.—Diagrams of the brain in each experiment, showing the number and size of gross petechial hemorrhages observed post mortem. In the first 5 experiments the left hemisphere, and in the last 5 experiments the right hemisphere, was irradiated.

and a generalized seizure occurred. All other animals ate on the first postoperative day but did not take a full day's diet. Another animal, cat 7, showed stiffness of the neck and generalized muscular rigidity on the first postoperative day. Meningitis was suspected. Penicillin, 1,000 units every four hours, was given intramuscularly for twenty-four hours, and fluids were administered subcutaneously. By the second postoperative day the animal was normal. By the third postoperative day all the animals seemed normal, and they were killed between the third and the fifth postoperative day.

POSTMORTEM OBSERVATIONS

Preparation of Brains.—The cats were killed with light ether anesthesia, followed by decapitation. In 5 cases the brain was removed immediately and a color

photograph taken within five minutes. The brain was then placed in dilute solution of formaldehyde U. S. P. and agitated for twenty-four hours. The brain, including the brain stem and cerebellum, was then sectioned coronally at 0.25 to 0.5 cm. intervals. In 1 case the brain was immediately photographed, sectioned and placed in various fixatives. In 2 cases the brain was perfused in situ by injection of dilute solution of formaldehyde U. S. P. (1:4). In 2 of the animals the brain was perfused with acacia U. S. P., according to the method described by Windle and associates.[13] Sections were then prepared in Windle's modified Nissl stain for vessels and nerve cells. The following stains were used routinely on selected blocks of half-hemispheres: The hematoxylin and Van Gieson stain; Nissl's stain and Windle's modification of the Nissl stain; phosphotungstic acid, hematoxylin and eosin; Laidlaw's connective tissue stain; the Loyez stain; gold chloride–mercury stain; silver carbonate impregnation for oligodendroglia; the benzidine stain;

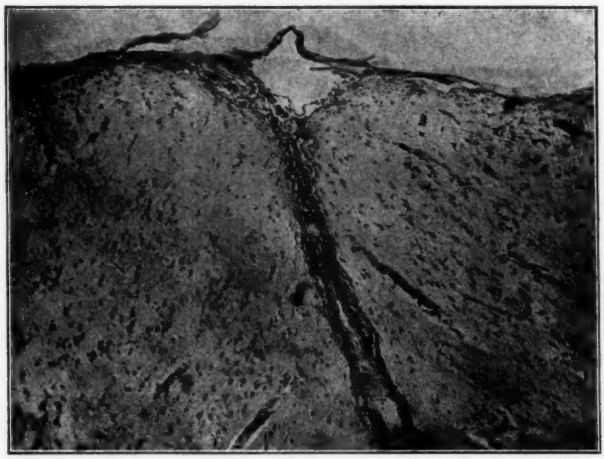

Fig. 10.—Hemispheral edema of two adjacent irradiated gyri. Note the obliteration of the intergyral fissure and the marked increase in the size of the perineuronal and the perivascular spaces. The cellular elements and vessels are molding in the direction of the traction. Hematoxylin–Van Gieson stain; low power magnification.

Spielmeyer's and Bielschowsky's methods, and stains for fat were also used routinely.

Gross Examination.—Postmortem examination of the unfixed and fixed brains showed considerably more swelling of the previously irradiated hemisphere (fig. 7). In most instances the swelling was so great that the impressions from the bony limitations were prominent on four sides. Usually these impressions were not found over the nonirradiated hemispheres. The gyri were flattened and the fissures almost obliterated on the exposed side. On the control side they

13. Windle, W. F.; Rhines, R., and Rankin, J.: Stain Technol. **18**:77-85, 1943.

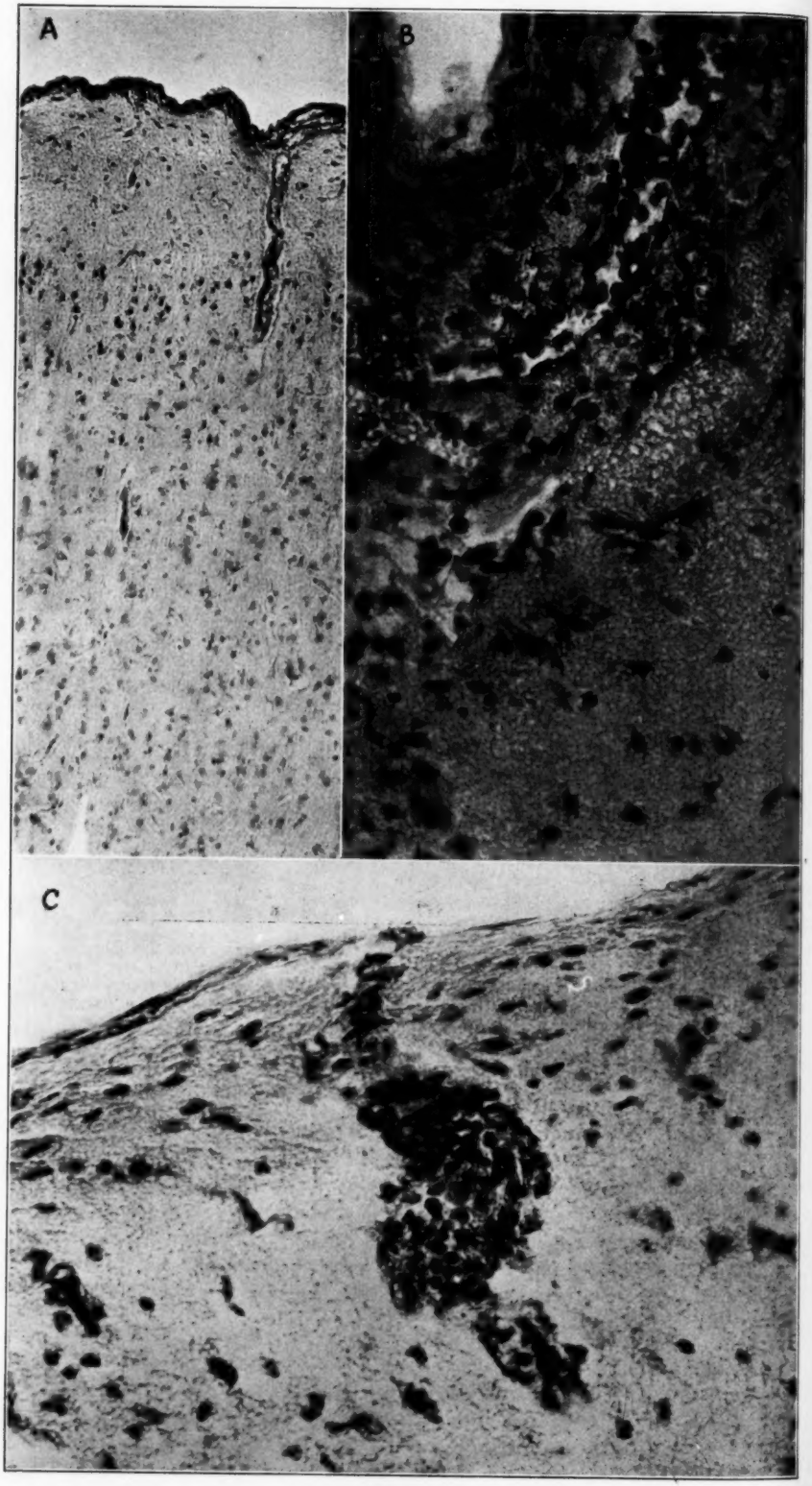

Figure 11

(See legend on opposite page)

were narrowed but always recognizable as distinct fissures. Diffuse edema of the white matter was more noticeable on the previously irradiated side. In each instance the lateral ventricle of the previously irradiated side showed a depression in the roof of the body at its middle and anterior portions (fig. 8). Adhesions between the temporal muscle or the dura and the brain were commoner on the previously irradiated side, being still commoner and denser on the fourth and fifth postoperative days than on the third postoperative day.

Multiple petechial hemorrhages were noted in the cortices of all the previously irradiated hemispheres (figs. 7, 8, 9, 12, 13 and 14). As many as sixteen discrete hemorrhages (cat 2) were seen over an irradiated hemisphere. In 1 cat, thirteen hemorrhages were already visible at the end of the five hours of exposure. In the remaining 9 cases none was visible during the exposures but they were seen at autopsy, three to five days later. The gyrus suprasylvius medius, the gyrus lateralis and the gyrus ectosylvius medius, in this order of frequency, bore most of the hemorrhages. These gyri receive ultraviolet radiations at approximate right angles to their surfaces. Four irradiated hemispheres showed gross subpial blood, apparently where numerous petechial hemorrhages had become confluent (fig. 7). Several others showed gross subpial blood over the convexities and within the fissures (*6* and *8* in figure 8).

Coronal sections of the brain showed that the petechial hemorrhages were largely within the cortex (fig. 8). In 2 experiments hemorrhagic cysts were noted within the white matter beneath cortical areas of rather diffuse hemorrhage (*6* in figure 8).

Histologic Examination.—Histologic examination of half-brain sections will be discussed under the separate headings. To describe all the changes following exposure of the brain to air is beyond the scope of this paper. The one hemisphere, which was exposed to ordinary air in each case, was used as a control for the preparations and description of the irradiated hemisphere. Only differences and exaggerations, which were found within the irradiated hemisphere, will be reported.

General Changes: The more directly irradiated gyri showed the more pronounced histologic changes. There was diffuse edema, resulting in traction of the blood vessels, nerve cells and glia (fig. 10). The

Fig. 11.—(*A*) Appearance of the perivascular spaces over the nonirradiated hemisphere. Note the absence of perivascular cellular infiltration. (*B*) Higher power magnification of advanced arteritis appearing in the meninges over an irradiated hemisphere. Note also the intense perivascular infiltration of polymorphonuclear and mononuclear cells, with infiltration of the plexiform layer. (*C*) Low power magnification of inflammatory change around a vessel in the plexiform layer within the gyrus suprasylvius medium of the irradiated hemisphere. Hematoxylin–Van Gieson stain.

perivascular and perineuronal spaces were widened in all instances, yet not remarkably more so than on the nonirradiated side (fig. 11*A*).

Changes in the Meninges: Limited sharply over the irradiated gyri was a rather prominent accumulation of inflammatory cells and serum (fig. 11*B*). The few cells on the nonirradiated side were largely mononuclear, while those on the irradiated side were largely polymorphonuclear. The meningeal vessels were engorged with blood, and in some instances the internal elastic membrane was fragmented. In 2 instances mitotic figures were identified in either an endothelial cell or a fibroblast of the pia-arachnoid on the irradiated side. There

Fig. 12 (cat 2).—Appearance of the brain three days after operation. Note the presence of numerous petechial hemorrhages over the convexity of the middle gyrus and the amount of exposure obtained. Compare with *2* in figure 9.

was fibrous thickening of the pia-arachnoid in the area exposed to the radiation.

Vascular Changes: Dilatation of meningeal, cortical and deeper vessels were more pronounced on the irradiated side. The meningeal and cortical vessels seemed most affected, showing swollen endothelial cells with a granular cytoplasm, a large nucleus, eccentric deposits of chromatin and, in 1 instance, a mitotic figure. An inflammatory cuffing of the meningeal and cortical vessels was observed (fig. 11*B* and *C*).

All irradiated hemispheres showed petechial hemorrhages (figs. 7, 8, 9, 12, 13, 14). These were largely limited to the cortex, but in

Fig. 13.—Relative size of a cortical petechial hemorrhage in the gyrus suprasylvius medius of an irradiated hemisphere. Benzidine stain; low power magnification.

Fig. 14.—Distribution of another petechial hemorrhage in the gyrus suprasylvius medius on the irradiated side. Benzidine stain; low power magnification.

50 per cent of the cases hemorrhages were observed in the white matter also. Diapedesis of red blood cells, both in the white and in the gray matter, was also a constant feature (fig. 15). There were no petechial hemorrhages on the nonirradiated side.

Ischemic patches were seen in the gray and the white matter of the irradiated hemispheres. Clear fluid–filled cysts were noted in the white matter in 3 cases (fig. 16*A* and *B*). In 1 instance a tiny

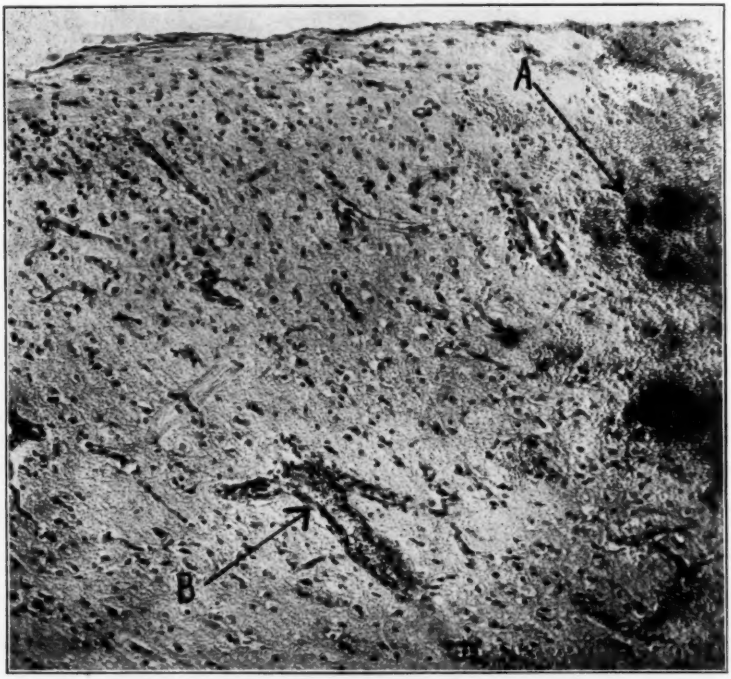

Fig. 15.—Appearance of the cortex following a five hours' exposure to ultraviolet radiation. Note the pronounced hemorrhages at *A*, the perivascular edema at *B* and the diapedesis of red blood cells throughout the superficial layers of the cortex.

cyst was found within the internal capsule. Each cyst was lined with numerous gitter cells, swollen astrocytes and oligodendrogliocytes, reactive microglial cells, a few mononuclear cells and an area of nerve cell destruction with numerous neuroglial fibers scattered in the immediate zone (fig. 16*B*). In 2 instances hemorrhagic cysts were observed in the gray matter, and in 1 other case a hemorrhagic cyst was noted within the white matter (cat 6, fig. 8). The nonirradiated

hemisphere failed to show cystic areas of any type, although ischemic areas in the gray and white matter were not uncommon.

Nerve Cell Changes: Chromatolysis was limited largely to the second, third and fourth layers of the irradiated cortex (fig. 17*B*). Central chromatolysis was commoner in the deeper layers and pyknotic changes commoner in the superficial layer. In many cells the dendrites

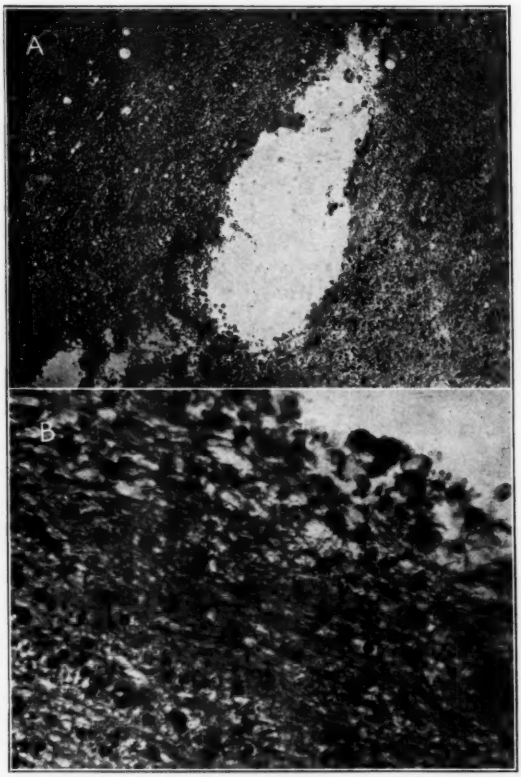

Fig. 16.—(*A*) Large cyst at the juncture of the gray and white matter in the gyrus lateralis of an irradiated hemisphere. Note multiple daughter cysts below and to the left and the presence of red blood cells at the lower left border of the large cyst. (*B*) High power magnification of the right border of the large cyst. Note the compound granular corpuscles and the swelling of the oligodendroglia. Hematoxylin–Van Gieson stain.

gave the impression of a corkscrew-like distortion. Their axis-cylinders were swollen, beaded and sometimes fragmented. In the third and fourth layers of the cortex the Nissl substance appeared in fine granular deposits in the periphery of the cell. In some instances the outer

protoplasm vanished completely, and clear spaces with curved limits appeared, which gave a serrated and fissured outline to the cell. The neurofibrils could not be demonstrated in the shrunken cells, while in the acutely swollen cells they were displaced to the periphery (fig. 18). In further advanced changes the neurofibrils were fragmented and formed short and fine argentophil granules. Although the disappearance of nerve cells is difficult to estimate, the long parallel extracellular fragments of neurofibrils in the Gros-Bielschowsky impreg-

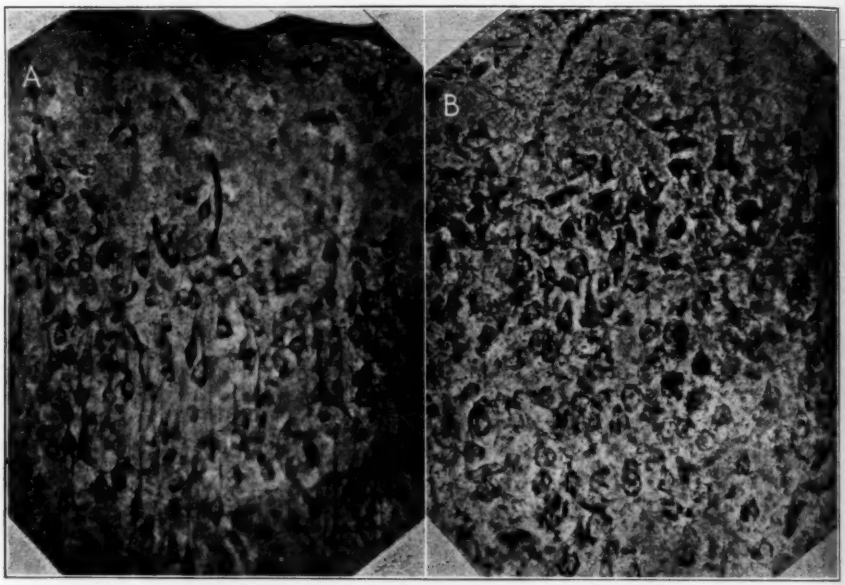

Fig. 17.—*A*, appearance of nerve cells in the gyrus suprasylvius medius of the nonirradiated side. *B*, appearance of nerve cells in the same area of the irradiated hemisphere. Note the large number of acutely swollen cells in the deeper layers, with the occasional shrunken cell in the more superficial layers. Many cells have lost their processes and show advanced degrees of chromatolysis, and a few have almost disappeared. Note the reaction of the microglia in the plexiform layer above. The silver impregnation (*B*) is of slightly higher magnification than is *A*.

nations suggested a rather marked disappearance of nerve cells on the irradiated side, by the fourth and fifth postoperative days (fig. 18).

Interstitial Cell Changes: The vascular attachments of large astrocytes on the irradiated side showed swelling and a rather loose approximation to the vessels. In the immediate zones surrounding cysts and hemorrhages there was early gliosis. When the gyrus was deformed by the marked edema, the astrocytes became elongated, and

their fibers were oriented in the direction of the migration of the gyrus. This mechanical alteration produced astrocytes which in some respects resembled classic pathologic pilonidal glia.

The oligodendroglia remained normal in both the nonirradiated and the irradiated hemispheres, except in areas, surrounding cysts and hemorrhages, where they showed acute swelling (fig. 16*B*).

The microglia showed acute ameboid forms in the plexiform layer of the irradiated hemisphere (fig. 17*B*). In some instances fat was demonstrated within a microglial cell at the periphery of the plexiform

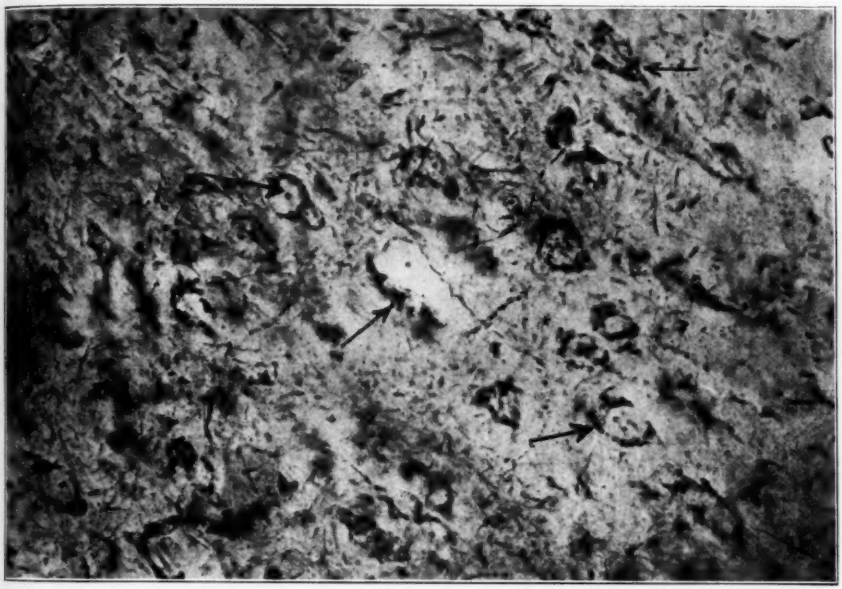

Fig. 18.—Appearance of nerve cells in the third cortical layer of an irradiated hemisphere. Note the almost complete disappearance of the center nerve cell, the fragmentation of the neurofibrils, the acutely swollen cells, the eccentric and swollen nuclei and the clumping and peripherally placed intracytoplasmic substance. Bielschowsky's stain for neurofibrils.

layer. Loss of lateral spines was common, giving the entire plexiform layer a dark granular appearance. In the layers of the pyramidal cells acute swelling was observed. Normal microglial cells were noted on the nonirradiated side.

Myelin: The myelin sheaths showed bulbous swelling in both the nonirradiated and the irradiated hemispheres. Fragmentation of the myelin was noted in the deeper white matter of the pyramidal radiations in only the irradiated hemispheres. Phagocytes, filled with an opalescent

orange hyaline substance, were found in the vessels and in the perivascular spaces nearby. Fat-filled phagocytes were demonstrated on both sides in the perivascular spaces and in the vessels, largely within the gyri which were exposed either to air or to ultraviolet light.

COMMENT

Blum's [9] résumé of the evidence that the erythema of sunburn is due to the action of a histamine-like dilator substance released by injured epidermal cells, which diffuses to the underlying minute vessels, suggests a similar mechanism for the dilatation of the cerebral vessels noted after short exposure to ultraviolet radiation. An increased concentration of dilator substance has actually been demonstrated in the skin [14] and in the blood of animals that have been exposed to ultraviolet radiation.[15]

Since essentially the same tissue responses occur after exposure to ultraviolet radiation as occur in inflammatory processes in general,[16] one may reason that cerebral edema, increased capillary permeability and migration of the leukocytes from the capillaries are from liberation of leukotoxine and necrosin,[17] elaborated as a result of cell injury inflicted by ultraviolet radiation.

Electroencephalograms demonstrated depression of cortical activity during the stage of vascular congestion, which was approximately one hour after exposure to ultraviolet radiation. In one record epileptiform spikes were observed. The animal had a clinical seizure at the end of five hours. The explanation for these abnormalities is not clear. It is suggested that they may be a result of the vascular disturbances, of the direct effect on ganglion cells or of a specific chemical disturbance. It is of interest that Prados and associates [12] found changes in the electrical activity of the cortex after simple operative exposure of the cat brain to air, but these workers had also obtained gross cerebral swelling.

The appearance of petechial hemorrhages between the time of operative closure and the postmortem examination, three to five days later, is evidence of irreversible damage to vessels. In 1 instance petechial hemorrhages were already visible over the exposed area toward the end of the operative exposure. In all cases petechiae were

14. Harkins, H. N.: Surgery **3**:430, 1938. Menkin, V. J.: Chemical Basis of Injury in Inflammation, Arch. Path. **36**:269-288 (Sept.) 1943. Taylor, F. H. L.; Levenson, S. M.; Davidson, C. S.; Adams, M. A., and MacDonald, H.: Science **97**:423, 1943.

15. Kolnitz, H.: Am. J. Physiol. **129**:399, 1940. Laurens, H., and Kolnitz, H.: M. Rec. **152**:209, 1940.

16. Blum, H. F.: Physiol. Rev. **25**:483-530, 1945.

17. Menkin, V.: J. Exper. Med. **64**:485, 1936.

observed post mortem. The majority of hemorrhages appeared over the more directly irradiated gyri. No petechial hemorrhages were observed in the nonirradiated hemispheres exposed to air. Prados and associates,[12] however, encountered hemorrhages on simple exposure of the cat brain to air, but, as previously stated, they also encountered gross swelling.

In our experiments, edema during exposure was difficult to recognize. It was suspected on some occasions on the irradiated side. On the control side, swelling was never definitely identified during exposure. However, all brains showed swelling at autopsy, which was greater on the irradiated side.

When the cerebral hemisphere is exposed to ultraviolet rays, an inflammatory type of reaction occurs. This consists of a serous and cellular exudate, mostly polymorphonuclear in type, with cuffiing of vessels, and dilatation of meningeal, cortical, and even deeper, vessels in the subjacent white matter with endothelial swelling and other evidence of arteritis. Thickening of the pia-arachnoid over the exposed surface occurs. Diapedesis and petechial hemorrhages are common in the gray and white matter of the irradiated hemisphere, while areas of relative ischemia and small cyst formation are sometimes seen. In view of the absence of such changes on the control side, it is reasonable to conclude that they are primarily due to the toxic effect of the ultraviolet radiations, and more specifically to their absorption within the cytoplasm and nucleus of the affected cells.

Neuronal alterations found within the irradiated hemispheres may be an effect of ischemia or of the direct absorption of ultraviolet radiations by the protoplasm of the nerve cells. Direct injurious effects seemed to predominate, for the maximal neuronal changes were seen in the more superficial layers of the cortex. The histologic work of Hamperl, Henschke and Schultze [18] demonstrated nuclear degeneration in the skin following exposure to ultraviolet radiations deep to the rather thick stratum corneum, which is a fairly good screen for ultraviolet light. Further resistance to sunburn is dependent on formation of melanin and thickening of the stratum corneum, with resulting increase in its screening action.[19] In the absence of such screening factors, the penetrating action of ultraviolet light may well reach the third layer of pyramidal cells. Pathologic alterations following exposure would suggest this depth of penetration.

18. Hamperl, H.; Henschke, U., and Schultze, R.: Naturwissenschaft. **27**:486-487, 1939.

19. Blum, H. F., and Kirby-Smith, J. S.: Science **96**:203-204, 1942. Kirby-Smith, J. S.; Blum, H. F., and Grady, H. G.: J. Nat. Cancer Inst. **2**:403-412, 1942.

Fibrous astrocytes exhibited possibly a slight degree of hyperplasia. Echlin [20] and Prados and associates [12] reported moderate hyperplasia of neuroglia on exposure of the cat brain to air alone, being more marked in the marginal zone in the area exposed.

SUMMARY AND CONCLUSIONS

The recent introduction of ultraviolet radiation for antibacterial purposes has led to its use on the exposed human brain during neurosurgical procedures. Since the need for antisepsis is more important in the longer neurosurgical procedures, the necessity of knowing the prolonged effect of radiation on the exposed animal brain seems to be of immediate importance.

Ten cats were subjected to bilateral craniotomies. Ultraviolet radiations, having 80 per cent of their wavelengths in the vicinity of 2,537 angstrom units, were allowed to project onto one hemisphere for five hours, while the other hemisphere, used as a control, was shielded from the radiations. Pathologic studies were made after the animals were killed, on the third, fourth or fifth postoperative day.

Exposure of the cat brain to ultraviolet radiation under actual human operating conditions produced dilation of meningeal and cortical vessels, usually within thirty to sixty minutes. Gross congestion subsided in twenty minutes by elimination of the ultraviolet radiation. Reirradiation, however, provoked a rapid redilation.

Petechial hemorrhages were observed on the irradiated side in all animals at autopsy, on the third or fifth postoperative day. In 1 animal cortical petechiae were already visible toward the end of the five hour exposure.

On inspection of the exposed cortex slight edema was suspected in only a few instances on the irradiated side. It was difficult to evaluate and recognize during exposure. However, in all the cases gross edema was observed at autopsy and was greater on the irradiated side.

Cortical electrograms were taken throughout the exposure in 4 animals. In 3 experiments depression of activity occurred after an exposure of one hour. In 1 animal epileptiform spikes developed after one hour, and this animal had a clinical seizure at the end of five hours. The electrographic alterations supervened during the stage of vascular congestion.

An intense inflammatory response was observed in the irradiated area. This consisted of vascular congestion of meningeal and cortical

20. Echlin, F. A.: Thesis, Faculty of Graduate Studies and Research, McGill University, Montreal, Canada, 1939.

vessels; transudation of serum; cellular exudate, with cuffing of vessels; diapedesis, and numerous petechial hemorrhages.

Within the irradiated hemisphere ganglion cells were reduced in number, showed swelling and disruption of cell membranes and occurred as pyknotic forms in the more superficial layers.

Fibrous astrocytes showed evidence of mild hyperplasia of their projections on the irradiated side. The oligodendroglia remained normal, except about cysts and hemorrhages, where acute swelling occurred. The microglia was reactive, especially in the plexiform layer over the irradiated hemisphere.

It is concluded that prolonged ultraviolet irradiation of the brain is harmful and that further investigation is necessary to establish how long the brain may be irradiated without showing irreversible pathologic effects. Physiologic and electrographic observations during ultraviolet irradiation of the exposed brain suggest that with frequent irrigation of the brain exposure of thirty to forty-five minutes will not result in permanent harmful effects.

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ELECTROENCEPHALOGRAPHIC CHANGES AFTER PREFRONTAL LOBOTOMY

With Particular Reference to the Effect of Lobotomy on Sleep Spindles

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THERE are relatively few reports on electroencephalographic changes following lobotomy.[1] Most reports [2] are in agreement that electroencephalographic slowing occurs after lobotomy, that it is most prominent anterior to the plane of incision and that the degree of slowing decreases within the first few months after operation. Electroencephalographic changes are not correlated with the psychiatric clinical condition,[1d] but may be correlated with seizure phenomena. An abnormal preoperative electroencephalogram increases the chances that a patient will have convulsions postoperatively,[1e] and the postoperative electroencephalographic abnormality may become more rather than less pronounced in patients in whom seizures develop.[1c] Cohn attributed the postoperative slowing to brain injury, whereas Stevens and Moscovich [1d] attribute it to the interruption of corticothalamocortical impulses.

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Aided by a grant from the Fluid Research and Teaching Fund of Yale University.

This study was undertaken in connection with the Connecticut Cooperative Lobotomy Research Program in collaboration with Dr. Burness Moore, of the Department of Psychiatry and Mental Hygiene of Yale University School of Medicine, and Dr. Benjamin Simon, of the Connecticut State Hospital.

1. (*a*) Davis, P. A.: Electroencephalographic Studies on Three Cases of Lobotomy, Psychosom. Med. **3**:38-50, 1941. (*b*) Hutton, E. L.; Fleming, G. W. T. H., and Fox, F.: Early Results of Prefrontal Leucotomy, Lancet **2**:3-6, 1941. (*c*) Cohn, R.: Electroencephalographic Study of Prefrontal Lobotomy: A Study of Focal Brain Injury, Arch. Neurol. & Psychiat. **53**:283-288 (April) 1945. (*d*) Stevens, H., and Moscovich, A.: Clinical and EEG Investigation of Prefrontal Lobotomy Patients, Am. J. Psychiat. **104**:2, 1947. (*e*) Greenblatt, M.; Levin, S.; Healey, M. E., and Solomon, H. C.: Effect of Prefrontal Lobotomy, Presented at the Annual Meeting of the American Electroencephalographic Society, Atlantic City, June 12, 1948.

2. Footnote 1, *b*, *c*, *d*, *e*.

Our interest in the effect of lobotomy on the electroencephalogram in human beings was aroused by the report of Morison and Bassett,[3] who describe bursts, trains or spindles of 8 to 12 per second waves in cats under pentothal sodium® anesthesia. Morison and Bassett investigated these spindles on the assumption that they were analogous to the 8 to 12 per second, or normal alpha, activity of waking human beings. The runs were found to originate in the thalamus, where they occurred spontaneously and independently of cortical or other afferent stimuli. The 8 to 12 per second runs disappeared after thalamic degeneration in chronically decorticate animals.

It seemed likely to us that the 8 to 12 per second spindles described by Morison and Bassett [3] are analogous to 12 to 15 per second spindles which occur in human beings in normal sleep,[4] rather than to the alpha waves of waking adults. Clark and Ward [5] reported that such runs occur in cat electroencephalograms in spontaneous and in barbiturate-induced sleep.

In order to test our assumption, pentothal sodium® was administered to patients by one of us (J. C.) before and at various intervals after lobotomy in order to determine what changes occur in the 12 to 15 per second sleep spindles after the corticothalamocortical fibers have been cut in human beings. Changes in the waking electroencephalogram after lobotomy are also reported.

METHOD AND MATERIAL

The subjects were 48 patients, aged 17 to 76, subjected to prefrontal lobotomy by the open technic, a modification of the Lyerly method. Operations were performed at the Grace–New Haven Community Hospital by Dr. William German and his staff and at the Connecticut State Hospital by Drs. William Scoville and Benjamin Whitcomb. One hundred and sixty-three electroencephalograms were obtained before and at intervals of one day to eighteen months after operation. In 8 instances, only preoperative electroencephalograms were obtained, and in 4 instances only postoperative recordings were available.

Electroencephalograms were recorded with a six channel Grass electroencephalograph. Routine recordings were obtained of frontal, central, occipital and temporal leads bilaterally taken with reference to interconnected grounded ear leads.

During 55 of the recordings, a 2.5 or 5 per cent solution of pentothal sodium® was injected intravenously. Pentothal recordings were usually obtained of anterior temporal, frontotemporal, prefrontal and frontal leads anterior to the burr holes and of central, posterior temporal and occipital leads posterior to the burr holes. Pentothal records were usually obtained on one side of the head only.

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3. Morison, R. S., and Bassett, D. L.: Electrical Activity of the Thalamus and Basal Ganglia in Decorticate Cats, J. Neurophysiol. **8**:309-314, 1945.

4. Loomis, A. L.; Harvey, E. N., and Hobart, G.: Distribution of Disturbance Patterns in the Human Electroencephalogram, with Special Reference to Sleep, J. Neurophysiol. **1**:413-430, 1938.

5. Clark, S. L., and Ward, J. W.: Electroencephalogram of Different Cortical Regions of Normal and Anesthetized Cats, J. Neurophysiol. **8**:99-112, 1945.

Preoperative records were classified according to the Gibbs[6] classificatory system. Postoperative abnormalities fell into the following groups in order of severity:

1. Records in which slowing is extreme in all leads but is most prominent frontally. One to 3 per second frequencies predominate in such records.

2. Records in which the slow wave abnormality, although usually diffuse, is definitely more prominent in a lead or leads on one side than on the other.

3. Records in which slowing involves the frontal and central leads, sometimes also the temporal leads.

4. Records in which slowing is confined to the frontal leads and varies from 1 to 6 per second.

5. Records in which slowing is confined to the frontal leads and consists of extremely slow (½ to 1 per second) base line sway with normal frequencies superimposed.

6. A normal electroencephalogram or a return to the preoperative electroencephalogram.

RESULTS

The effects of lobotomy on the waking electroencephalogram will be discussed first.

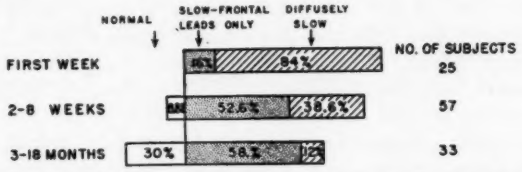

Fig. 1.—Degree of electroencephalographic abnormality found at various intervals after prefrontal lobotomy.

Immediate Effects of Lobotomy on the Electroencephalogram.—Fourteen electroencephalograms were obtained on the first or second postoperative day. All but 1 showed extreme slowing most prominent in the frontal but present in all leads, and 3 showed, in addition, much more abnormality on one side than on the other. This degree of abnormality persisted for variable periods: In 1 patient it was present for six weeks, and in 1 patient it was not present at two days. The incidence of this degree of abnormality decreased gradually during the first four postoperative weeks. From three to eighteen months after operation, only 12 per cent of patients showed this degree of abnormality.

Intermediate and Prolonged Effects of Lobotomy on the Electroencephalogram.—The degree and extent of electroencephalographic slowing decreased as the time interval after lobotomy increased (fig. 1).

6. Gibbs, F. A.; Gibbs, E. L., and Lennox, W. G.: Electroencephalographic Classification of Epileptic Patients and Control Subjects, Arch. Neurol. & Psychiat. **50**:111-128 (Aug.) 1943.

The change was gradual for the group as a whole. Serial electroencephalograms were taken once or twice a week on 15 patients, and it could be seen that in individual patients the degree and extent of electroencephalographic slowing decreased abruptly. In some patients this abrupt decrease in abnormality occurred within the first post-

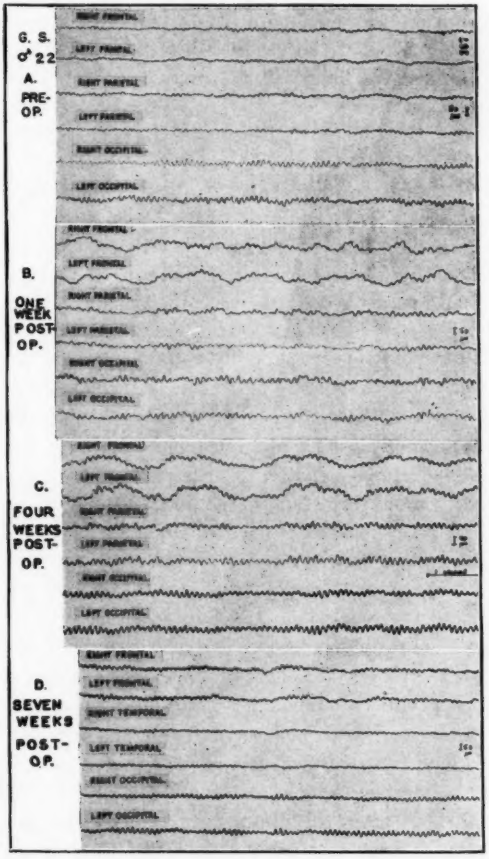

Fig. 2.—Characteristic example of electroencephalographic changes after lobotomy.

operative week; in some it was delayed to the fourth to the sixth postoperative week. When the decrease in the degree of abnormality occurred, it typically occupied only a week or two. Gradual stepwise return to a base line level, occupying four to five weeks, was the exception, occurring in only 3 patients. There was little change

between electroencephalograms taken at three and at twelve to eighteen months. In 2 patients, there was an exacerbation of electroencephalographic abnormality at one and two weeks respectively, with later return toward normal.

The changes in a fairly characteristic case are illustrated in figure 2. The patient had a normal preoperative electroencephalogram (fig. 2 *A*). At seven days after lobotomy, both frontal leads show extreme slowing with very little normal activity (fig. 2 *B*). At four weeks, marked frontal slowing persists, but normal frequencies are superimposed on the frontal slow waves (fig. 2 *C*). At seven weeks, the frontal leads show predominantly normal activity, at the preoperative rate, with very little if any baseline sway (fig. 2 *D*).

Only a few patients showed slowing of other than the frontal leads; slowing was almost always confined to the leads situated in the frontal lobes anterior to the plane of incision. In the few patients in whom slowing involved the central leads, posterior to the plane of incision, it was felt that operative trauma was responsible. The final result in the majority of patients was slowing confined to the frontal leads, usually consisting of a very slow base line sway on which normal frequencies were superimposed (fig. 2 *C*). We have not encountered this type of abnormality in any condition except prefrontal lobotomy.

Electroencephalographic Changes During Pentothal Sodium® Anesthesia Before and After Lobotomy.—Electroencephalographic changes during the intravenous injection of pentothal sodium® are dependent on the dose and rate of injection. Slow injection is accompanied with marked increase in fast activity, as described by Brazier and Finesinger.[7] In our experience, patients receiving injections in this manner regain consciousness quickly when the needle is withdrawn and no sleep spindles appear. In these patients, the average dose was 0.38 Gm. injected in three minutes and fifty-four seconds. Faster injection of larger amounts produces immediate high amplitude, swinging, slow waves, 2 to 4 per second, with little or no preceding fast activity. Sometimes these immediate slow waves are intermixed with transient 12 to 15 per second waves, which are unaffected by lobotomy. If the needle is withdrawn after the appearance of slow waves, the patient usually continues in what appears to be normal sleep for ten to fifteen minutes and can then be aroused easily. After five to ten minutes of this normal sleep, 12 to 15 per second waves sometimes appear intermittently. In these patients, the average dose was 0.44 Gm. injected in two minutes and thirty seconds. The 12 to 15 per second waves may appear in a short or a long series; there may be only a

7. Brazier, M. A. B., and Finesinger, J. K.: Action of Barbiturates on the Cerebral Cortex, Arch. Neurol. & Psychiat. **53**:51-58 (Jan.) 1945.

few such sequences or they may recur repeatedly; they may occur with a flat base line, with or without a crescendo-decrescendo appearance, or they may appear on the ascending or descending portion of a slow wave (examples in figure 3). These variations in appearance make it difficult to find a term which is accurately descriptive. The terms "bursts," "trains," "runs," "sequences of 12 to 15 per second

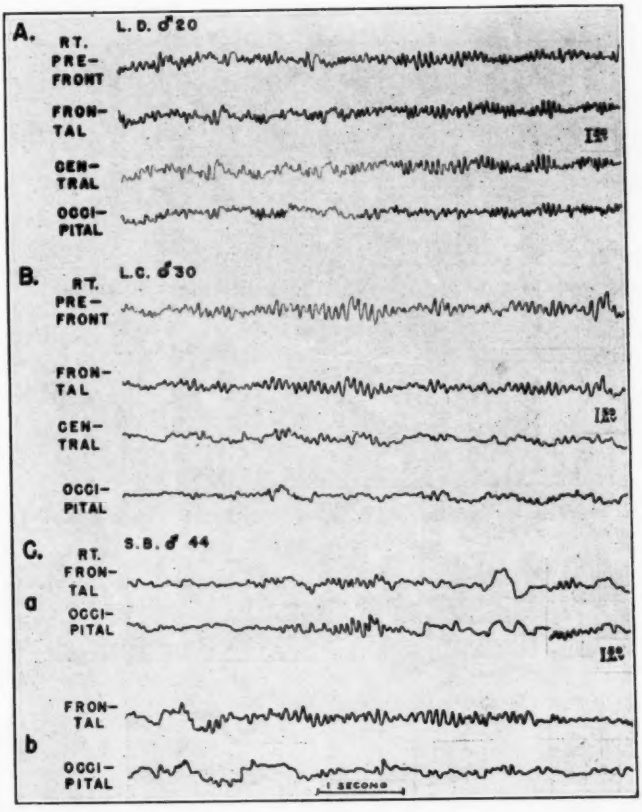

Fig. 3.—Runs of 12 and 12 to 14 per second waves in pentothal sodium*-induced sleep before lobotomy.

waves" and "sleep spindles" have been used. "Runs of 12 to 15 per second waves in sleep," although clumsy, is the most accurately descriptive phrase, in our opinion, and will be used in this communication.

In reviewing the pentothal records, it was noticed that runs of 12 per second waves in sleep differ from runs of 14 to 15 per second

waves in sleep both in prelobotomy and in postlobotomy recordings. Runs of 12 per second or 14 to 15 per second waves in sleep occurred in 23 of the 25 prelobotomy electroencephalograms. However, it was rare for both 12 and 14 to 15 per second sleep runs to occur in the same person (5 cases) (example in figure 3 *C*), and 12 per second runs were encountered more commonly (10 cases) than 14 per second runs (7 cases).

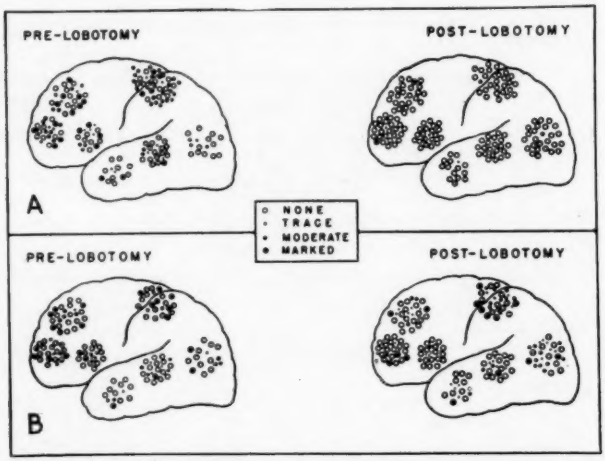

Fig. 4.—*A*, distribution of 12 per second spindles; *B*, distribution of 14 to 15 per second spindles.

Prelobotomy and Postlobotomy Distribution of Runs of 12 per Second and 14 to 15 per Second Waves in Sleep with Respect to Frontal (F) Leads Anterior to the Plane of Incision and Central (C) Leads Posterior to the Plane of Incision

	F Only or F > C	F = C	C > F	C Only	None	Total
12/sec.						
Prelobotomy	9 (36%)	5 (20%)	0	1 (4%)	10 (40%)	25
Postlobotomy	1 (3%)	0	1 (3%)	1 (3%)	27 (90%)	30
14/sec.						
Prelobotomy	1 (4%)	7 (28%)	3 (12%)	1 (4%)	13 (52%)	25
Postlobotomy	2 (7%)	4 (13%)	6 (20%)	11 (37%)	7 (23%)	30

Head areas involved also tend to differ (fig. 4). Although both 12 and 14 to 15 per second sleep runs usually appeared in both frontal and central leads before lobotomy, there was a tendency for 12 per second runs in sleep to appear most prominently, and occasionally only, in the frontal leads (fig. 3 *B*); whereas the runs of 14 to 15 per second waves in sleep did not occur only in the frontal leads, tended to be most prominent in the central leads and occasionally appeared prominently in the occipital leads (fig. 3 *A*). There was no significant difference in the temporal leads.

Finally, runs of 12 per second differ from runs of 14 to 15 per second waves in sleep in their response to lobotomy (fig. 4). Of 30 postoperative pentothal electroencephalograms, runs of 12 per second waves appeared in sleep in only 3 (the table). Two occurred in the first two postoperative days, and 12 per second runs were absent in 1 of these patients on a repeat electroencephalogram with pentothal

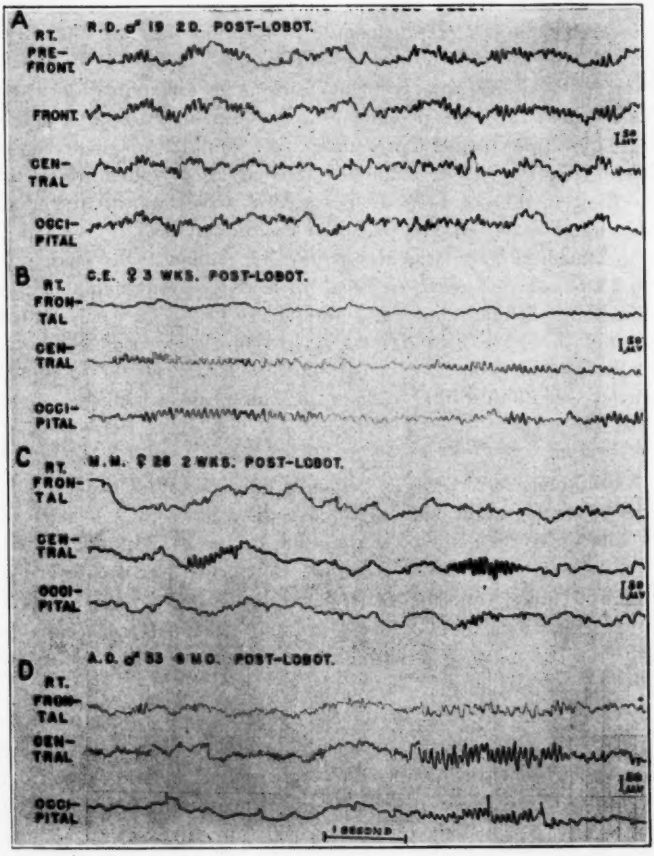

Fig. 5.—Runs of 14 to 15 per second waves in pentothal sodium®-induced sleep after lobotomy.

sodium® two weeks later. One patient had runs of 13 per second waves in sleep in the central leads only both preoperatively and three weeks postlobotomy.

By contrast, runs of 14 to 15 per second waves in sleep occurred in 23 of 30 postlobotomy pentothal records (the table).

After lobotomy, the 14 to 15 per second runs occur less frequently anterior to the plane of incision and more frequently posterior to the plane of incision than before lobotomy. Preoperatively, runs of 14 to 15 per second waves in sleep occurred in 12 patients, and were present only posterior to the (future) plane of incision in 1. Postoperatively, runs of 14 to 15 per second waves in sleep occurred in 23 patients and appeared only posterior to the plane of incision in eleven (the table).

Several examples of postlobotomy sleep spindles are illustrated in figure 5. In the first example in this figure (fig. 5 *A*), 14 to 15 per second runs in sleep appear in all leads after lobotomy. Fourteen to 15 per second runs are prominent in the central leads after lobotomy but are only questionably present in the frontal leads in examples *C* and *D* of figure 5. In example *B* of figure 5, taken in pentothal-induced sleep, runs of 14 to 15 per second waves appear in the central and occipital leads but are absent in the frontal lead.

Particular interest attaches to the 11 patients who had runs of 14 to 15 per second waves in sleep in leads posterior but not anterior to the plane of incision. The waking record of 1 such patient is illustrated in figure 2, and it is typical of the records of the other patients in this group. Normal alpha activity, at the preoperative frequency, occurs in frontal leads in the waking records of these patients.

COMMENT

Our findings agree in general with those of other observers [2] that electroencephalographic slowing occurs after lobotomy, is most prominent anterior to the plane of incision and decreases in extent and degree with increasing time after operation. That the electroencephalogram eventually returns to normal in 30 per cent of cases agrees with findings in other conditions, notably head injury.[8] The electroencephalogram usually returns to normal within three months in spite of gross and irreversible brain damage.

In other conditions, at least,[9] the electroencephalogram either does not return to normal or again reverts to abnormality if brain damage is followed by seizures. Since there are only 3 patients in our group in whom seizures have developed after operation, we are not able to draw any conclusions on this score, but the work of others [1c, 8]

8. Williams, D.: The Electroencephalogram in Chronic Post-Traumatic States, J. Neurol. & Psychiat. **4**:131, 1941.

9. Roseman, E., and Woodhall, B.: The Electro-Encephalogram in War Wounds of the Brain; with Particular Reference to Post-Traumatic Epilepsy, A. Research Nerv. & Ment. Dis., Proc. **25**:200-219, 1946. Gibbs, F. A., and Gibbs, E. L.: The Electroencephalogram in Encephalitis, Arch. Neurol. & Psychiat. **58**:184-192 (Aug.) 1947.

suggests that the same generalization applies to lobotomy as to head injury and encephalitis.

Other investigators have not mentioned specifically the type of abnormality which is the commonest residuum of lobotomy in our series. This abnormality is confined to leads placed anterior to the plane of incision and consists in an extremely slow base line sway (½ to 1 per second) with normal frequencies superimposed. This abnormality cannot be attributed to trauma, since it does not occur in patients with other types of injury. It occurs only in patients after prefrontal lobotomy in our experience. For this reason we ascribe it to the severance of fiber tracts between the cortex and the thalamus but are at a loss to explain how such an electrical effect is produced.

Some of the patients in our series had transient slowing in leads posterior as well as anterior to the plane of incision, and both clinical and electroencephalographic findings suggested that there was operative trauma in the ordinary sense in these patients.

On 56 occasions, pentothal sodium® was given intravenously. When the dose was high and the time of administration short, the electroencephalogram showed immediate, swinging, 2 to 4 per second waves with little or none of the initial fast activity which occurs with slow injection.[7] When initial slowing occurs, the patients are apt to sleep normally and can be aroused easily after ten to fifteen minutes. Usually after five to ten minutes of sleep, runs ("bursts," "trains" or "spindles") of 12 to 15 per second waves appear. Before lobotomy, 12 per second runs are somewhat commoner and tend to be distributed more anteriorly than 14 to 15 per second runs. After lobotomy, with very few exception (2 of the 3 exceptions occurred within the first two postoperative days), runs of 12 per second waves in sleep do not appear in any lead. This corresponds with the experimental findings of Morison and Bassett [8] and 8 to 12 per second pentothal spindles do not appear in chronic decorticate cats, in which autopsy shows degeneration of thalamic nuclei, especially centralis medialis and lateralis. In cats, evidence points to the thalamus as the site of origin of "sleep spindles." [8] Our findings in human subjects conform with experimental findings and suggest that in human beings, as in cats, disappearance of 12 per second runs in sleep is due to degeneration of thalamic nuclei. In view of the report by Freeman [10] that nucleus centralis medialis shows the most marked degeneration after lobotomy, it seems likely that it is this thalamic nucleus which is the site of origin of runs of 12 per second waves in sleep.

Runs of 14 to 15 per second waves in sleep posterior to the plane of incision are more prominent after than before lobotomy. Presumably

10. Freeman, W., and Watts, J. W.: Retrograde Degeneration of the Thalamus Following Prefrontal Lobotomy, J. Comp. Neurol. **86**:1, 1947.

they arise in the nucleus centralis lateralis, which is spared after lobotomy.[10] Runs of 14 to 15 per second waves in sleep may appear anterior to the plane of incision after lobotomy, but they occur much less often than before operation. This would appear to indicate that in many patients enough thalamocortical fibers are spared after operation to allow conduction. Particular interest attaches to those postoperative subjects who have prominent runs of 14 to 15 per second waves in sleep in the central leads but none in the frontal leads; one must assume that in these patients the thalamocortical fibers have been severed completely. (It is, of course, possible that fibers to the orbital surface of the frontal lobe have been spared, since this area is inaccessible to scalp electrodes.) The waking records of these patients show normal alpha activity anterior to the plane of incision. It would appear, then, that normal regular alpha activity is not dependent solely on intact thalamocortical connections. The theory appears unlikely that the normal alpha rhythm is dependent for its maintenance on intact corticothalamocortical reverberating circuits.

It appears evident that 8 to 12 per second sleep spindles in cats are analogous to sleep spindles in human beings and not to the human waking alpha rhythm as postulated by Morison and Bassett.[3]

Finally, this report amply confirms the finding of Ulett[11] in dogs and of Cress and Gibbs[12] in human beings that absence of sleep spindles is of localizing value.

SUMMARY AND CONCLUSIONS

1. Patients with lobotomy have generalized slowing, most prominent in the frontal leads, in the first few postoperative days. Slowing decreases in degree and extent slowly in the group, abruptly in individuals, and there is little change after three months.

2. The commonest long term electroencephalographic residuum of lobotomy consists in ½ to 1 per second base line sway with normal frequencies superimposed.

3. Runs of 12 per second waves in sleep are most prominent in anterior leads before operation and virtually do not occur after lobotomy. It is assmued that they originate in the nucleus centralis medialis of the thalamus and that their absence in all leads after operation is due to degeneration of this nucleus.

4. Runs of 14 to 15 per second waves in sleep are most prominent in central leads before lobotomy, are more frequent in central leads

11. Ulett, G.: Electroencephalogram of Dogs with Experimental Space-Occupying Lesions, Arch. Neurol. & Psychiat. **54**:141-149, 1945.

12. Cress, C. H., and Gibbs, E. L.: Electroencephalographic Asymmetry During Sleep in Patients with Vascular and Traumatic Hemiplegia, to be published.

and are less frequent in frontal leads after than before lobotomy. It is assumed that they originate in the nucleus centralis lateralis of the thalamus. When they are still present anterior to the lobotomy incision, it must be assumed that severance of fiber tracts between the cortex and thalamus is incomplete. When they are absent in frontal leads and present in central leads after lobotomy, it must be assumed that thalamofrontal fibers are completely severed (unless orbital fibers have been spared).

5. Persistence of normal alpha activity in the frontal leads of patients whose sleep records demonstrate complete severance of corticothalamic fibers casts serious doubt on the theory that alpha activity is maintained solely by thalamocorticothalamic reverberating circuits.

Miss Olga Noto and Miss Ann McCorkindale gave technical assistance in this study.

THE PHOBIC SYNDROME

A Study of Eighty-Six Patients with Phobic Reactions

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A STATISTICAL study of my records reveals that in the course of treating 3,100 psychoneurotic patients, 86 with a severe phobic syndrome have been encountered. The incidence of patients with phobia in my practice is approximately 2.5 per cent of all psychoneurotic persons. The phobic syndrome occurred twice as often in women as in men. The majority of those seeking treatment were under 40 years of age; 2 were under 20, and there were a few with the chronic phobic syndrome between 40 and 50 years of age. Roughly, 75 per cent of these phobic patients had one or more seriously neurotic parents; in some instances the parents were phobic. The number of siblings and the patient's position in the family constellation appeared to play no part in the picture, and over half of them had three or more siblings. Very few were emotionally close to any sibling, indicating that phobic persons do not form numerous close personal attachments. Eighty-five per cent had been married, 4 more than once; only 2 had been divorced. In general they had few children. The majority had a semblance of an occupation but had never worked very hard, and most of them had been only moderately successful in their undertakings, chiefly because they were handicapped by fear. These 86 phobic patients were outstanding as to intelligence, and came from financially secure families who were well able to give them a good education. One third had failed to take advantage of their opportunity, mainly because of homesickness when they were sent away to school. The majority had been reared in an overprivileged environment. Uniformly, they lived poorly balanced lives, were remarkably free of bad habits, but lacked sensible, and even usual, self regimentation. They were dependent people, without hobbies, who seldom took part in competitive games and practically never participated actively in any sport which might be considered mildly dangerous. They desired to be social, but feared and avoided social situations, in spite of the fact that they could nearly all make an unusually good impression and were superficially likable. They were not particularly satisfactory marital partners, nor very good parents. They were likely to neglect marital and parental obligations, putting most

of the burden on spouse or governess. In spite of this, their spouses loved them, probably because of their easy-going, pleasant personalities.

Sex did not seem to play an important part in the phobic syndrome, regardless of freudian literature to the contrary. These patients showed only mild, but apparently sufficient, interest in sex. Several open-minded and competent staff psychiatrists made a systematic study to uncover unusual sexual frustration, guilt and inhibitions occurring in childhood or later. The results of their study did not demonstrate any significant connection between sexual maladjustment and phobic reactions.

The majority of these patients had been suffering from phobias for years before seeking treatment. When they came to me, most of the 86 patients were untreated, but some had seen other psychiatrists, and a few had been psychoanalyzed, without benefit.

The phobic syndrome is apparently one which arises in young adults when an apprehensive, dependent, emotionally immature person is trying to realize his ambitions and become an independent, successful member of society. These patients were, without exception, extremely apprehensive, cyclothymic, unusually pleasant personalities. Many of them had experienced recurring mild depressions and mild elations. In some instances the specific phobias began during a depression. The phobias were always made worse by the recurrence of a depression but, if untreated, never disappeared during a mild elation. In a large number of cases these patients were living under environmental conditions which were extremely unsatisfactory if their desires, ambitions, basic natures and personalities were taken into consideration. In many instances environmental conditions had to be altered in order to effect a permanent cure.

It is significant that over 90 per cent of these phobic patients had unusually good health; only 7 of the 86 had ever had a serious physical illness. The physical examinations revealed an essentially normal condition except that all of them were physically "soft" and most of them showed evidence of an overactive vegetative nervous system. The absence of somatic illness is worth noting. The phobic person is one who has been overprotected, brought up "soft." Perhaps if he had had a severe physical illness, he might have been toughened by that experience.

Not 1 of these 86 patients with a phobic syndrome ever acquired a psychosis. A number had used alcohol and/or barbiturates to relieve their discomfort, but none had become seriously addicted to either.

REVIEW OF MEDICAL LITERATURE

The phobias are generally classified under the heading of anxiety hysteria. The term "anxiety reaction, phobic syndrome," is more

descriptive and is preferable. A review of the literature (which is nearly altogether freudian analytic) reveals elaborate explanations based on the analysis of a few cases. It would seem that in some instances these theories have been accepted without adequate scientific verification and then elaborated on. There are several conflicting theories. The subject of phobias has not been thoroughly and scientifically studied.

The following statements summarize the most important material in the literature:

This neurosis occurs in infantile persons. It is a process of regression to childish dependence on omnipotent adults for protection.

Phobias are due to dynamic interplay between Oedipus situations and pregenital fixations.

Claustrophobia represents a compromise between independent strivings and the regressive pull back to mother.

Sexual repression by the parent on whom the child had fixation and dependence gave rise to the aggression-fear complex plus feelings of guilt, and neurotic symptoms result.

The phobia is a surface manifestation of underlying anxiety, a substitute for the real problem.

Janet classified the phobias under psychasthenia. He stated that greater sexual activity, more social life, opportunity for fuller self expression and learning to give and take are of importance in treatment. He stated the belief that many of these patients live under an unfavorable environment and that to get them permanently well the environment must frequently be modified. The favorable effect of modification of environment we have found to be true in a significant number of our cases.

The freudian analytic literature states that phobias are the result of sexual frustration, unconsciously feared or desired punishment, traumatic experiences, conditioned reactions or unfavorable emotional conflicts with near relatives. The fears themselves are considered substitutes for repressed complexes. As described in the medical literature, the psychodynamics and treatment of the phobias are in the main not convincing.

PSYCHODYNAMICS OF THE PHOBIC SYNDROME

This study of 86 patients indicates that there is a simple explanation of this condition and that the phobic syndrome is a well defined entity. These fundamentally apprehensive persons, with cyclothymic tendencies, have hyperactive vegetative nervous systems that cause unpleasant physical symptoms which they do not understand, and which thus give rise to further fear. Constitutionally, phobic persons are unusually suggestible, imaginative and sensitive. They have considerable drive, high standards of conduct, an exceedingly active intelligence and much ambition. They have been reared "soft," usually by neurotic parents, on

one of whom they are still emotionally dependent. They reach maturity handicapped by fundamental natures which they have not learned to use to advantage, and by the way in which they have been reared. They attempt to take part in a competitive world without adequate psychologic preparation for living. They come in contact with the dangerous realities of existence, suffer frustration and experience unpleasant physical symptoms; thus becoming unfavorably conditioned to specific situations, they regress to a childish level of adjustment. They subconsciously use their specific fears as an excuse for not attaining success.

Although the literature lists one hundred and seven specific phobias, all having Greek names, such as claustrophobia, zoophobia and nyctophobia, they are all similar in both origin and treatment. Since the phobic patients exhibit a characteristic clinical picture, startlingly alike in most details, there is a question whether there is sufficient justification for subdividing the phobias into these various types, describing them by one symptom, an outstanding fear, only. The present report deals with the usual phobic picture, the phobic syndrome in which fear occurs in a greatly exaggerated form. Any person who is afflicted with unreasonable, persistent, specific handicapping fears has phobias. Phobias may be for place, space or time. When a person has evolved a reaction pattern of always evading situations in which these specific fears might arise, he is phobic. Patients who suffer from the phobic syndrome are afraid to venture into the world alone, and when they do they feel the need of a protective influence. They carefully avoid crowds and all social gatherings, as well as open spaces, such as streets and country roads, where they might be entirely alone. They refuse to enter any place from which they cannot escape quickly and with dignity. They refuse to attend parties; go to restaurants, ride on trains, elevators, subways, airplanes or ships, or go to beauty parlors or barber shops. Some such persons refuse to enter theaters, schools, churches, stores, offices or factories or to attend lectures and concerts. They particularly dread towns, tunnels and islands. They fear experiencing the disagreeable symptoms of faintness, weakness, fatigue, palpitation, perspiration, nausea, tremor and difficulty in breathing which have become associated with certain situations or conditions, and so avoid any place in which these unpleasant feelings may occur. They dwell morbidly on the possibility that they may experience paralysis of thought, loss of memory, depression or insanity. The phobic person is well aware of all the situations which he avoids, but at first he tells the doctor only a few of his fears. Rationalization or deception has become such an important part of his life that he will deceive even the physician whom he consults for treatment. The psychodynamics of the phobic syndrome are indicated in these 3 abbreviated typical case histories.

CASE 1.—A woman aged 41, married for twenty years, had two children. The mother was neurotic; the patient was afraid of her father and was close to her mother and dependent on her.

The patient had been a sensitive, timid, shy, self-conscious child. She had attended private school and one year of college but did not study. She came from a privileged home, with a protected Southern environment of culture, but she had never made any effort to secure additional cultural knowledge. She was desirous of position and the good things in life but made no effort to acquire them. She had been born into a friendly environment but made no close friends herself. She was afraid of failure in social situations. At the age of 21 she married a man she had grown up with. He traveled much, and she stayed with her parents in his absence. She worried about her two children and tried to evidence deep affection by constantly expressed oversolicitude.

The active phobic syndrome was precipitated by the death of her mother; by her moving to New York, which she thought strange and unfriendly, and where she felt that she was a failure, and had an ectopic gestation. During recovery from the operation for this pregnancy, while she was shopping in New York, she became weak and thought she might die. The situation was exaggerated by ignorance and mishandling by the policeman, hotel clerk, husband and doctor. Likewise, her illness served to keep her husband (who intended leaving that day on a business trip) at home. Similar episodes occurred during the next ten years, at the end of which period treatment began. The patient was then chronically anxious and feared death or sudden illness if she went on the street, on trains, in cars or to the theater or church. She had reached a point at which she could not perform any of her duties and was helpless. Her husband had to remain at home with her, and even then she continued to be frightened. She was in good physical condition except for being overweight and "soft."

Recovery followed two months of treatment. Five years have now passed. She takes a completely different attitude toward life and is a successful wife and mother. After her recovery she persuaded her husband to change his conservative attitude and has helped him to rise from a $12,000 a year position to the presidency of his company, at $30,000. She carries active social responsibilities, which help him immeasurably.

CASE 2.—A woman aged 27, married three years, had one child. Both parents were phobic, the patient being strongly attached to her father. She came of an overprivileged, overprotective home. She attended a small, noncompetitive school, then a boarding school for three years and college for four years. She was an average student. She never tried very hard to do anything, and never had to; she was always "spoon fed." She felt, with truth, that she had never accomplished anything; fear and laziness held her back.

The patient was intelligent, sensitive, cyclothymic and severely and constantly apprehensive. She had few adult interests; she was ambitious but easy going and socially attractive. She had married three years before, when her husband was in service; she traveled around with him. After the birth of her baby, she returned to her parents' home, to live in luxury, without responsibilities.

Her phobic syndrome was activated by her husband's discharge from service, her moving from her own home to a town where she knew no one and his frequent absence on business, together with the knowledge that she was failing as a wife, mother and citizen.

While walking down the street alone (her husband being away), she had a sudden attack of palpitation, cold sweat, weakness, nausea, dizziness and fear of losing control of herself. Thereafter, she was afraid to go out alone or to go into situations unaccompanied if she could not readily escape. She became so upset that she had to go home to her father.

After seven weeks of treatment the condition was relieved; the patient returned home and assumed her responsibilities adequately. She now leaves her parents entirely alone, takes an active part in community life, skiis and has learned to fly a plane.

Case 3.—A man, aged 56, had been married twice. His first wife had been mentally ill and was a suicide; he was separated from his second wife, who was also a mental patient; he had three children.

The patient had an unhappy home life as a child; his parents fought constantly; his mother died of melancholia. He was brought up in luxury and was overprotected. He attended private schools; he was never a good student and took no part in competitive sports. His father lost his money during the patient's adolescence, and the patient became terrified and afraid of poverty. He felt under great pressure to be a success. The patient had been apprehensive all his life; he was suggestible, sensitive, intelligent, ambitious. He had become aware of his fears in childhood and tried to conceal them as being unmanly. A self-made man, he had worked hard, acquiring no interests outside of work. He was pleasant, given to mild depressions and always anxious about the future. He had few close friends, although he would have liked to have many. His sex life had been satisfactory before marriage and with both wives. He had had no extramarital sexual affairs. He had no feelings of guilt or undue inhibitions about sex.

He had a lifelong tendency to be hypochondriacal but had had no previous neurotic break. His illness was precipitated by unsettled and unsatisfactory business conditions; his partner was a crook, who almost wrecked the firm. He was unhappy about his second wife and had a feeling of guilt that perhaps he should not have refused to continue to live with her. He feared that he might not be able to rear the children successfully.

While living with this background of past and immediate fear of failure as a human being, his friend died in his office, practically in his arms, of angina pectoris. (The patient's father had died of angina pectoris, a disease which the patient thereafter feared.)

He became frightened about his condition and had pains in the chest, fear of heart disease, loss of appetite, indigestion, eructation of gas, weakness, salivation and insomnia. He was afraid to go to his office and afraid to be alone; he stayed at home, would not travel by train or car and avoided friends. This condition continued for eight months; then the patient went to live in a hospital and would not walk out of sight of the hospital.

When accepted for treatment, the patient was seriously underweight, had tremor of the hands, was "soft" and had a greatly overactive vegetative nervous system.

The patient was treated intensively for seven weeks and was entirely relieved of symptoms. He resumed business, traveled abroad and reorganized his life, with more play, hobbies, interests and friends. He moved from city to country, from which he commuted to work. Since his treatment in 1937, a period of over ten years, he has gone through several emotional strains without recurrence of symptoms or any neurotic manifestations.

TREATMENT

The phobic syndrome could probably be prevented by recognizing and treating potential phobias in childhood. Most persons with phobias start battling fear at an early age. Some children are basically apprehensive, while others are not fundamentally anxious. Apprehensive children should be recognized as such, the earlier the better, and trained in the art of dealing with fear by discounting it rather than by denying or fighting it. To offset their dread of fear, they must be taught to enjoy the pleasures and rewards of an adventurous life.

Neurotic parents, particularly if they themselves have phobic personalities, should realize that so far as their apprehensive children are concerned they are psychologically infectious. They should seek treatment and continue with it until they are cured. Only then will they learn not to overprotect their children or allow them to become emotionally dependent. Apprehensive children need to be loved, not as household pets, but as respected human beings. Well-to-do people should see to it that their children are not brought up under hot-house conditions or protected from too much hardship, but that instead they are taught that this world is no place for a luxury-loving, apprehensive, dependent person. While growing up, children should be taught to endure normal hardships gracefully. This in no sense implies that any child will thrive under a Spartan regime. He should, however, learn from early years to meet each situation as it arises; to handle problems encountered at each age level in a manner compatible with his understanding and ability at that time. No issues need be forced on him; daily life brings a sufficient number of opportunities for practice in social adaptation, but he must not be protected from ordinary difficulties, as in learning how to handle them he strengthens his moral fiber. The apprehensive child should be taught to take an active part in sports, to swim, dive, sail, ski, shoot and fish; to visit away from home and parents at an early age; to travel alone, speak in public, dance, act—in short, to know that the thrill of being afraid and daring to do in spite of fear is one of man's greatest and most continuously rewarding experiences.

For the adult whose phobias have carried over from childhood, gaining strength with each year until they have matured into a phobic syndrome that is dominating his life, psychologic reeducation through brief psychotherapy, including participation in group activities, is indicated.

Plan of Therapy.—The phobic person needs to be socialized. The best results are accomplished with the patient temporarily away from home, living in a favorable environment, associating with a group of twenty or thirty pleasant people and taking part in their social activities.

Simple reassurance that the patient is not in danger is of no avail. The patient already knows this, is ashamed of being dominated by fear and yet alone cannot overcome it. Likewise, admonition that he should, and persuasion that he can, overcome his fear do little lasting good. If the patient is depressed as well as phobic, the depression must be successfully treated before the physician attempts to alleviate the phobic syndrome.

The process of psychologic ventilation, in which the patient discusses his mental conflicts, is seldom in itself curative. The patient may ventilate for months. At times, in order that he may not have to grapple with his specific fears, he may even manufacture new conflicts to keep the doctor satisfied. It has been said that discovering and removing emotional conflicts will cure phobias. This is seldom true, since the phobic patient suffers from extreme emotional immaturity and specific conditioned reactions. The patient should at first be encouraged to discuss his emotional difficulties, since part of his illness derives from emotional dyscrasias. Through such discussion the patient establishes an emotional rapport with the doctor and attains a better understanding of himself and his problems. All of this will be useful during the later treatment. This process of mental catharsis should be guided by an experienced physician who understands the personality and training of the phobic patient and who, with this knowledge, so directs the analysis of personality that the patient may quickly comprehend the nature of his illness.

Phobic patients profit by a course of mental hygiene whereby they may learn to use and have faith in their intelligence. The information so gained helps restore their confidence, gives them mental tools to employ and teaches them the principles of personal mental hygiene. This course of psychologic reeducation should describe the physiology of the vegetative nervous system and explain how it gives rise to physical symptoms. It must emphasize the role of the attention in accentuating symptoms, explain the nature of emotions, describe conditioned emotional responses and teach the patient how to deal with his emotions. Psychologic reeducation must include a practical and appealing philosophy of life, emphasizing that human beings must have as guiding and impelling forces both objective and idealistic purposes in life.

While undergoing treatment, these patients should live on a daily schedule. This should include work, rest, exercise and play, best carried out in a group. The schedule should include an hour's walk twice daily, at first with people, then alone. Exercise and athletic games will harden the patient physically, help him to take pride in his physical condition and reassure him about his physical efficiency.

Exercise will likewise reduce some of the unpleasant reactions caused by an overactive vegetative nervous system. Daily cold baths on arising are helpful both physiologically and psychologically. After the first week of treatment, which has included a daily hour with the psychiatrist, it is suggested that soon the patient will be ready to attempt some of the things which he has previously avoided. In three or four weeks the patient is usually ready to attempt to recondition one of the simple situations of which he is afraid.

Treatment thus far has served the purpose of (1) establishing a rapport between patient and doctor; (2) affording the patient an opportunity to ventilate his conflicts, and assisting him to discover what these really are; (3) giving him an understanding of his own nature and personality; (4) teaching him the essentials of personal mental hygiene; (5) socializing him to some extent; (6) building up confidence in his physical condition and decreasing his tendency to experience vegetative symptoms, while at the same time increasing his tolerance toward these manifestations.

The patient is now ready for the second phase of his treatment, namely, the reconditioning of his specific fears—a process which is already in progress, even though the patient has been unaware of it. He is now mixing socially with a group of people, is accustomed to walking several miles daily and is less afraid of physical symptoms. These facts are called to his attention as a means of further increasing his confidence in himself and in the doctor.

The following principles are involved in the process of reconditioning specific fears:

1. The patient must be willing to accept temporary discomfort for the sake of earning future relief and rewards.

2. The patient must understand the principle of reconditioning his unpleasantly conditioned emotional reactions.

3. The patient must cooperate in specific undertakings that involve placing himself in situations of which he is afraid. For example, after he has several times walked a country road with others, he takes this walk alone. To divert his attention from himself and his fears, he is given an objective project—he makes a rough but fairly accurate map of the road he travels, indicating houses, lakes and side roads. When he has successfully accomplished this task, his confidence is greatly increased. He is then assisted to take a short train trip alone, with "doctor's orders" to make certain definite purchases while in town, these purchases involving considerable planning en route. Next he is to take an elderly relative or friend to church. Then he is instructed to go to a moving picture, to keep notes and to bring the doctor a written résumé of the plot, with names and descriptions of the actors and actresses. Later, he makes a trip to the city, perhaps to a famous restaurant for an unusually good luncheon, and then to an amusing matinée.

In each instance he has employed not his emotions but his intelligence. He cannot fail to realize that he can think and act wisely, regardless of how he may feel. Like thousands of other neurotic patients, he has learned to discount fear.

Specific phobias are best reconditioned in this order: walking alone, and going to movies, restaurants, beauty parlors and the theater. Lastly, the physician treats and removes whatever fears remain to be reconditioned. When the patient has once accomplished the things of which he is afraid, he must be put through the reconditioning process repeatedly and never again be allowed to avoid the things and situations from which he has been escaping. When a specific task is set by the doctor to help the patient recondition a fear, the patient must undertake it at once; he must not be allowed time to think it over. On completing the prescribed task, the patient reports at once to the doctor, in person. If the patient fails to carry out the project prescribed, little is made of his failure; but in the very near future a similar, and more interesting, task is set for him by the doctor, without the patient's being told in advance what it is to be.

A patient is not cured until none of his phobias is active. Most patients have a particularly powerful phobia which they do not at first mention to the doctor voluntarily; this must be discovered and eliminated. The patient must be warned that never, in any circumstances, can he obey a phobia; that if he does so he runs the risk—indeed the certainty—of again suffering from the phobic syndrome.

The third phase of treatment has been proceeding along with the first and second phases, namely, helping the patient to mature. In order that this may be accomplished, there must be a psychologic divorce between the patient and the parent or parents to whom he is emotionally attached. He must learn true independence, be taught to make his own decisions and to carry his full share of responsibility. He should travel, participate in adventurous sports and take an active part in business, philanthropy, community affairs and social life. If the patient has been living under unfavorable environmental conditions, he must be removed from these, or else the conditions must be radically altered.

Phobic persons are usually highly intelligent; they have initiative and ideas; they will be successful in whatever they undertake if only they can be persuaded to undertake any one of the many things well within their capacity. Reeducation teaches them to understand that their conflicts are due to immaturity and fear of failure versus an unusually strong desire for success. This approach gives them more confidence in their own ability. As they overcome their former phobic reactions, they enjoy the rewards of a successful, interesting life.

The fourth phase of therapy consists of supervising the patient for several months, encouraging and inspiring him to travel the road he has undertaken. Persons with phobias must be taught to develop and use inner resources, and the physician must see to it that all source of profit from a neurotic way of life is removed.

RESULTS OF TREATMENT

In reporting the results of treatment, there is always the danger that the reporting physician will be too optimistic; therefore, it is well that such reports be viewed with skepticism. Suffice it to say that the 86 patients in this study have been followed constantly over the years, and the following conclusions seem justified:

	No. of Patients
Permanently relieved from phobias	58 (67%)
Great improvement, patient working and living efficiently	21 (24%)
Slight improvement or no improvement	7 (9%)

In studying these case records, it is interesting to see that the personality and ability of the physician played a great part in securing results. Although all the staff psychiatrists used the same technics and approach, the brilliant, optimistic, confident, friendly doctor, with a light touch, got by far the best results. Thus, again, it is demonstrated that the trusted physician is the bridge over which the patient crosses to find faith and confidence in himself and in the world.

SUMMARY

This report on 86 patients with phobic reactions indicates that the phobic person is fundamentally apprehensive, with an overactive sympathetic nervous system and a cyclothymic makeup. He is usually too closely attached to one parent and has not entered into mature adult relationships with other persons, even though he may have married and become a parent. Having been brought up "soft," he reacts to the dangers of everyday living by acquiring protective phobic reactions. His fears are merely symptoms of an underlying neurotic state; therefore the treatment of an adult with phobias consists in emotional retraining and reconditioning of specific fears through psychologic reeducation or brief psychotherapy. In order that he may reap the rewards to which his exceptionally good personality entitles him, a potentially valuable but immature person must be assisted to grow up, to assume an adult's place in society. To bring this about, he must be taught to accept no orders from unreasonable fears.

Psychologic reeducation requires approximately two months of intensive treatment, which should be followed by several months of medical supervision to make sure that the patient does not regress into phobic or other neurotic reactions. This follow-up treatment is important, since phobic patients have a tendency to mislead the physician in an attempt to escape further treatment by making the doctor think that they are more nearly well than they actually are. The physician must be sufficiently experienced not to be deceived by such a person, who is frequently a master in deception. As long as any phobia is active, the patient is not cured.

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COMPARISON OF ADJUNCT GROUP THERAPY WITH INDIVIDUAL PSYCHOTHERAPY

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MOST physicians who have used group psychotherapy are enthusiastic about it as a therapeutic tool. Although much has been written on the subject of group therapy in the past thirty years,[1] very few writers have attempted to analyze their results statistically. Schilder [2] and Bierer [3] are exceptions. There are good reasons for this omission, and they apply to all attempts to analyze results from psychotherapy. Psychotherapy, in spite of the fact that it is on a more scientific basis now than ever before, still remains a highly subtle process, and each therapeutist develops his own individual skills. These do not lend themselves to ready analysis. Furthermore, the evaluation of therapeutic results is also a highly individual process. Results tabulated by one therapeutist are always open to serious doubt by others who do not know the exact criteria used to evaluate the results. What one person may call a cure another would call only a symptomatic cure. What one may call a permanent personality improvement, another may think of as merely a temporary change based on an unanalyzed transference relationship. These difficulties are inherent in any effort to analyze therapeutic results and diminish the value of any attempt to do so.

However, each psychiatrist learns by experience to evaluate his patients in his own mind, and his evaluations contain a certain amount of validity for himself; he can classify his results into categories, such as improved or unimproved, and the series will have a certain amount of internal consistency. Inasmuch as I have treated a fairly large series of patients over a period of several years, using individual psychotherapy and group psychotherapy, it seemed reasonable to try to

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Published with the approval of the Chief Medical Director, Veterans Administration, Washington, D. C., who assumes no responsibility for the opinions or views expressed herein.

1. Slavson, S. R., and Myers, G.: Bibliography on Group Therapy, New York, American Group Therapy Association, 1946.

2. Schilder, P.: Results and Problems of Group Psychotherapy in Severe Neuroses, Ment. Hyg. **23**:87 (Jan.) 1939.

3. Bierer, J.: Group Psychotherapy, Brit. M. J. **1**:214 (Feb. 14) 1942.

compare the results. I had used group therapy on a rather extensive scale for two years in an overseas Army general hospital, and I was impressed with the therapeutic value of group psychotherapy, although under war conditions any accurate scientific check of results was out of the question, since patients were lost track of after they left the hospital and no follow-up was possible. Also, the pressure of work was so heavy that attempts to keep accurate records of the work done was very difficult. These drawbacks are not present in an outpatient psychiatric clinic in which similar types of patients are studied and treated. Patients in this clinic have been accurately followed up, some for a year and a half. Complete records are available on each patient treated.

Group therapy has taken various forms.[4] Much experimentation was done in the group therapy used in this series of patients, but it took the form of testing different intervals of time between treatments and of grouping various types of patients together. In general, there was no change in the type of group therapy given. It consisted of analytic-interpretative therapy.[4] The groups were always small and informal, the patients talking and asking questions at will. There was never any attempt to make the meetings didactic or inspirational. In this series, group therapy was used entirely as an adjunct to individual therapy. None of the patients received only group therapy.

I was impressed with the seeming good results from group therapy and wanted to compare those patients receiving only individual psychotherapy with those receiving both individual and group therapy to see whether there was really a great difference, as seemed probable from the clinical impression. At the time the comparison was made, 153 patients had received group therapy. This was roughly 20 per cent of all patients who had been through the clinic. The files were searched for the records of all patients who had received individual psychotherapy without group treatment for use as a control series. Only 123 of these were available. Another 515 patients had received no treatment for various reasons. The control series included many patients who were not comparable with those in the series receiving group therapy, and these were therefore removed. They were persons with schizoid personality, schizophrenia in remission, paranoia, etc. The remaining control series were patients who could have and ordinarily would have received group treatment except for practical reasons. They included all female patients (group treatment not available for these), Negroes (for the same reason), persons from out of town, persons who could not get away from work, etc. Some 22 per cent

4. Thomas, G. W.: Group Psychotherapy, Psychosom. Med. **5**:166 (April) 1943.

of these, furthermore, showed a decided resistance to attending the groups. These too were eliminated, leaving a control group of 68.

Each patient was analyzed from various points of view. It was obviously impossible to analyze them in terms of length of treatment or number of hours spent in treatment, as this varied considerably. Some patients have been under uninterrupted treatment since the opening of the clinic two years ago. Others were treated for a relatively short time. Some have been seen at frequent intervals; others, infrequently. However, it was possible to analyze each patient as to whether he came regularly or was irregular in his attendance; also as to whether he had had more than a few psychotherapeutic interviews. Each person was analyzed in terms of whether he was improved or unimproved. Improvement was divided into five different categories, and a patient was not considered improved unless there was advance in at least three of these. These categories consisted of (1) relationship with the physician; (2) improvement in pathologic attitudes; (3) improvement in symptoms; (4) improvement in adjustment and (5) improvement in insight. In the group who had received group therapy as an adjunct there were 153 patients, of whom 66 per cent were classified as improved. In the group who received only individual psychotherapy there were 68, with 51 per cent improved. This is a difference of 15 per cent. It is interesting to note that in the 153 patients who received group psychotherapy only 3 became psychotic in the course of treatment, whereas in the original control group of 123 2 became psychotic. This seems like a negligible number in each group, and in only 1 instance could I feel that the therapy precipitated the psychosis.

Table 1 gives a comparison of results according to diagnosis. The new diagnostic categories of the American Psychiatric Association, as modified by the Veterans Administration, were used. For each patient a single diagnosis was made, based only on the outstanding mechanisms of his difficulty; hence there is no overlapping in the diagnostic categories. I do not question that this procedure is open to grave criticism. Per cents are given only in categories in which enough patients were treated to make these of any statistical value. The diagnostic categories are listed in descending order according to frequency. Of especial note are the good results obtained with the adjunct group treatment of obsessive-compulsive states. A further report will be written on this subject.

Table 2 shows the number and per cent of patients showing improvement in the different categories studied. Although patients receiving group therapy showed improvement in each category, it should be noted that the improvements in adjustment, insight and pathologic attitudes are marked.

Further analyses show that of 79 patients receiving adjunct group therapy, 51 per cent were regular in attendance at treatment sessions, whereas of 46 patients not receiving group therapy 68 per cent were regular in attendance. On the other hand, those with added group treatment had less tendency to break off treatment prematurely; of 65 patients receiving group therapy 42 per cent made more than a few

TABLE 1.—*Analysis of Results by Diagnostic Categories*

Diagnosis	Receiving Group Therapy			Not Receiving Group Therapy		
		Improved			Improved	
	Total Number	Number	Per-centage	Total Number	Number	Per-centage
Anxiety reaction	50	35	70	12	7	58
Conversion reaction	55	35	64	30	15	50
Obsessive-compulsive reaction	13	12	92	3	2	..
Epilepsy	12	8	66	2	0	..
Depressive reaction	2	1	..	3	2	..
Psychopathic personality	6	1	17	5	3	..
Sex deviate reaction	2	2	..	5	3	..
Schizoid personality	2	2	..	1	0	..
Manic-depressive reaction (in remission)	1	0	..	0	0	..
Encephalopathy, traumatic	1	1	..	2	1	..
Phobic reaction	2	2	..	1	0	..
Schizophrenic reaction	3	1	..	0	0	..
Somatization reaction	2	1	..	2	1	..
Alcoholism	2	0	..	2	1	..
Totals	153	101	66	68	35	51

TABLE 2.—*Analysis of Results by Categories of Improvement*

Category	Receiving Group Therapy		Not Receiving Group Therapy	
	Number Improved	Percentage	Number Improved	Percentage
Insight	85	55	24	35
Adjustment	99	64	35	51
Symptoms	92	60	33	49
Pathologic attitudes	97	63	26	38
Relationship with physician	126	89	44	65

visits to the clinic, and of 25 patients not receiving group therapy only 37 per cent made more than a few visits.

A final comparison shows that of 51 combat veterans receiving group therapy 33 per cent were improved, whereas of 12 not receiving such treatment only 18 per cent were improved. This would seem to indicate that combat veterans consistently do better when treated with adjunct group therapy.

SUMMARY

A total of 153 patients treated in a Veterans Administration mental hygiene clinic over a period of two years, using group therapy as an

adjunct, were compared with a total of 68 who received only individual psychotherapy. There was a difference of 15 per cent improvement in those who received group treatment as an adjunct.

The number of patients who became psychotic was negligible in both series.

The groups are compared by diagnosis and the improvement in those receiving group treatment was sometimes astonishingly larger than those not receiving this.

The series are compared as to the categories of improvement. Patients who received group treatment tend to develop better insight and adjustment and to change their pathologic attitudes to a greater extent than those receiving only individual treatment. Combat veterans especially seemed to do better with group treatment.

ROLE OF REPEATED TRAUMA BY PNEUMATIC DRILL IN PRODUCTION OF AMYOTROPHIC LATERAL SCLEROSIS

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THE ISSUE of trauma in the production or precipitation of amyotrophic lateral sclerosis is difficult to assess, as it is in all nervous diseases. Experience with 2 patients in whom symptoms of the disease appeared to follow the use of a pneumatic drill raised the problem of whether repeated trauma might play a role in precipitation of the disease, and for this reason we report the cases in question.

REPORT OF CASES

CASE 1.—*Amyotrophic lateral sclerosis in a 63 year old man who had worked with a pneumatic hand drill for nine months, symptoms appearing in the eighth month of its use; signs of anterior horn cell disease confined largely to the cervical portion of the spinal cord.*

History.—E. H., a 63 year old Armenian man, was admitted to the Jefferson Hospital on May 5, 1947, with the complaint of weakness and twitching of the arms. Twenty months before entrance he began to work with a pneumatic hand drill, which he steadied against his shoulder in cleaning boilers. The tool was heavy enough to necessitate constant tension of his arm and shoulder girdle muscles in order to maintain it in position, and its action was forceful enough to shake his body. He had worked with this drill for nine months, and eight months after using it, or twelve months before entrance into the hospital, he began to be aware of the symptoms for which he sought relief. He had not worked with the drill for eleven months before entrance.

At the time of onset of his symptoms he noticed that his right hand was weak, to the extent that he was unable to carry objects in this hand or even to turn a lock with it. Two months later his left hand became similarly affected, and thereafter progressive weakness of both arms developed. For the two months before entrance he had noticed twitching and jumping of the muscles of his hands, arms and chest. For two and one half months he had been aware of emotional lability, irritability and hoarseness. He had had no pain.

Examination.—Tests revealed muscle atrophy, moderate in degree, including particularly the muscles of both shoulder girdles, arms, forearms and hands. Associated with this was muscle weakness, involving the affected muscles. Muscle fasciculations were noted in the deltoid muscles and, to a lesser degree, in the

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other muscles of the shoulder girdles, arms, forearms and small hand muscles. There was no evidence of atrophy or fasciculations in the muscles supplied by the cranial nerves, and no bulbar signs were noted. The biceps, triceps, radial, patellar and achilles reflexes were overactive, those of the left arm being faster than the right. Ankle clonus and pathologic toe signs were not elicited. The abdominal reflexes were decreased on the left side. The neurologic findings were normal in all other details.

Laboratory Data.—Blood studies revealed the following values: erythrocytes, 4,000,000; leukocytes, 6,000; hemoglobin, 71 per cent. Urinalysis gave entirely normal values on several occasions. The Wassermann reaction of the blood was negative. The fasting blood sugar was 74 mg. per hundred cubic centimeters. Spinal fluid studies revealed a pressure of 90 mm. of water; 1 cell per cubic millimeter; a negative Wassermann reaction on two occasions; colloidal gold curve 0000000000; a total protein of 48 mg. and normal Queckenstedt responses. A roentgenogram of the chest showed no abnormalities. Roentgenograms of the spine revealed moderate degenerative hypertrophic changes in the cervical vertebrae, with narrowing of the intervertebral disks between the fourth and fifth and the fifth and sixth cervical vertebrae. Oblique views of the cervical region of the spine revealed normal intervertebral foramens. Mirror laryngoscopy revealed normal motility and tension of both arytenoids and vocal cords.

CASE 2.—*Amyotrophic lateral sclerosis in a 50 year old man who had worked for over three years with a pneumatic drill; anterior horn cell disease pronounced in cervical region but present elsewhere.*

History.—W. P., a 50 year old Polish man, entered the Jefferson Hospital on Jan. 14, 1947, with the complaints of weakness and twitching of the muscles of both limbs and occasional transient numbness of the fingers.

He had worked in a bakery for two years (1940 to 1942) lifting barrels of flour, bearing the weight of the barrel on the left hand. From the summer of 1942 until December 1945 he had used a pneumatic drill and a pneumatic grinder in a defense job, the weight of the drill being supported by the left hand and arm. In December 1945 he became aware of fasciculations in the left side of the chest in the posterior axillary line. Three months later (February 1946) his left arm became weak and he noticed fasciculations in this limb as well. Eleven months later (January 1947), at the time of his admission to the hospital, both arms had become weak. He had had gonorrhea in 1936, but apart from this, the past history was noncontributory.

Examination.—Vitiligo was noted on the upper arms and in the axillas. The left side of the palate was weak, the palate moving to the right on phonation. The patient had atrophy of the hand and forearm muscles, and slight atrophy of the shoulder girdle muscles. The muscles of the thighs and legs were normal in bulk. The hand grips were very weak. Coarse fasciculations were seen in the pectoral, shoulder girdle, biceps and triceps groups, with occasional fasciculations in the thigh muscles. The biceps, triceps, radial and patellar reflexes were overactive; the abdominal reflexes were absent. No pathologic reflexes were elicited. The neurologic findings were normal in all other respects.

Laboratory Data.—Studies revealed the following: erythrocytes, 4,500,000; leukocytes, 9,000, and hemoglobin, 84 per cent. Urinalysis gave normal values on five occasions. The Wassermann reaction of the blood was negative. Spinal fluid studies revealed the following data: 145 mm. pressure; 1 cell per cubic millimeter; negative Wassermann reaction; colloidal gold of 0000000000; a total protein

content of 30 mg. per hundred cubic centimeters, and normal Queckenstedt responses. The serum bilirubin was 0.2 mg. The hippuric acid excretion test gave 4.86 Gm. in four hours. Roentgenograms of the cervical portion of the spine revealed no abnormalities. No platybasia could be demonstrated. Roentgenograms of the chest showed normal conditions. Gastric analysis with histamine revealed a peak of 6 units of free acid and 20 units of total acid.

COMMENT

The role of trauma in the production or precipitation of amyotrophic lateral sclerosis is a matter of considerable controversy. An exhaustive survey of the recorded cases of amyotrophic lateral sclerosis by Jelliffe [1] reveals no cases with trauma similar to the cases here recorded. Whether the repeated hammering of a pneumatic drill in our cases was sufficient to produce the amyotrophic syndrome may well be a matter of controversy, but it is valuable to speculate whether such trauma could be responsible for precipitating the symptoms of amyotrophic lateral sclerosis. If such a connection could be demonstrated, it would certainly be applicable to relatively few cases of the disease. The two cases here reported had in common the use of a pneumatic hand drill in working, and in both the patients after varying periods of time became aware of symptoms of anterior horn cell disease, such as weakness and muscle twitchings. In case 1 symptoms of this nature were noted eight months after use of a pneumatic drill, and in case 2 such a drill had been used for over three years. Many instances of amyotrophic lateral sclerosis develop, of course, without exposure to such hazards, but this does not detract from the interest aroused by our cases. The mechanism responsible for the production of the anterior horn cell disease is hard to define. It seems possible that repeated concussions could be transmitted to the spinal cord to produce changes in susceptible anterior horn cells. The possibility of involvement of the anterior horn cells by spinal cord concussion has been illustrated in a case recorded by Hassin,[2] in which an amyotrophic lateral sclerosis picture developed after spinal cord concussion. Incomplete efforts to produce experimentally selective disease of the anterior horn cells have been recorded by a number of observers. Schmaus [3] produced circumscribed foci of softening, cavities and system degeneration in the spinal cord by spinal cord concussion which was produced by repeated blows delivered to

1. Jelliffe, S. E.: The Amyotrophic Lateral Sclerosis Syndrome and Trauma, J. Nerv. & Ment. Dis. **82**:415 (Oct.); 532 (Nov.) 1935.

2. Hassin, G. B.: Concussion of the Spinal Cord: A Case with the Clinical Picture of Amyotrophic Lateral Sclerosis, Arch. Neurol. & Psychiat. **10**:194 (Aug.) 1923.

3. Schmaus, H.: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung, Virchows Arch. f. path. Anat. **122**:326 and 470, 1890.

a board attached to the backs of guinea pigs and rabbits vertically suspended. Slow, progressive paralysis resulted. Lhermitte[4] produced degeneration of the anterior horn cells of the spinal cord in a dog, resulting in progressive weakness and paralysis, by striking a number of stout blows against a board firmly attached to the dog's back. The anterior horn cells were shrunken and degenerated at autopsy several months after the experiment was carried out. Similar experiments were carried out by Stcherbak,[5] who applied a strong electric vibrator of the type used in vibrator massage in man to the right knee of rabbits. In 3 animals receiving two, five and six vibratory sessions with a total of five, forty-five and fifty-five minutes of applications respectively, the microscopic examination of the spinal cord showed nothing of significance. In 4 other rabbits microscopic examination of the spinal cord revealed changes of varying degree. 1. Vibration to the right knee for fifteen minutes consecutively resulted in extravasation of blood in the meninges, anterior horns, central canal and posterior horns of the lumbar area. The hemorrhages showed a preference for the gray matter and were most pronounced in the fifth and sixth lumbar segments. 2. Vibration applied during six sessions for five to fifteen minutes over an eight day period revealed dilatation of the central canal; necrotic cavities surrounding the central canal, as well as in the commissure, and the anterior horns; rarefaction of the tissue surrounding the necrotic areas with swollen axis-cylinders in the affected areas; swelling of the nerve cells of the anterior horns. The changes were localized in the fourth, fifth and sixth lumbar segments. The cervical and thoracic cords were found to be normal. 3. Total vibration of two hours and twenty-five minutes applied over a period of eight days was found to be associated with similar changes of a more advanced degree, the lesions being confined to the gray matter. 4. Total vibration of one hour over a seven day period revealed numerous areas of necrosis in the fifth and sixth lumbar segments, the changes being most pronounced around the central canal and in the base of the posterior horns.

A further basis is found in the occurrence of vasospastic phenomena in the hands in workers with pneumatic hammers. Similar Raynaud phenomena are found to occur in pianists and typists. The rapid percussion in the case of pneumatic hammers presumably sensitizes the digital vessels so that Raynaud symptoms occur in stone cutters who use a pneumatic hammer.[6] These are transitory and leave no serious

4. Lhermitte, J.: Spinal Amyotrophies Secondary to Trauma, Gaz. d'hôp. **103**:873, 1930.

5. Stcherbak, A.: Changes in the Rabbit Spinal Cord Due to Intensive Vibration, Encéphale **3**:521 (May) 1907.

6. Hardgrove, M. A. F., and Barker, N. W.: Vasospastic Disturbance of the Hands in Stone Cutters, Proc. Staff Meet., Mayo Clin. **8**:345 (June 7) 1933.

secondary effects, but they recur and seem directly traceable to the use of the pneumatic tool. Though no observations are available on the effect of such percussion on the vessels of the spinal cord, it seems not unreasonable to assume that they may occur in the use of pneumatic drills supported against the shoulder.

SUMMARY

In 2 cases of amyotrophic lateral sclerosis there appeared to be a direct relation between the use of a pneumatic drill and the onset of symptoms. The mechanism of production of symptoms may be related to vasomotor influences. The cases assume some importance from this standpoint rather than from that of direct relationship between trauma and the development of amyotrophic lateral sclerosis.

OBSERVATIONS ON "MAJOR" AND "MINOR" CAUSALGIA

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IN THE recent war, as in other wars, many young men received wounds of the extremities and injuries to the large peripheral nerves. Once more, as a result of such injuries, causalgia has appeared on a large scale. This painful condition of an extremity was known to Denmark [1] (1813) and Paget [2] (1864), before the classic and detailed description of the condition was given in 1864 by Mitchell, Morehouse and Keen [3] during the American Civil War. The term causalgia, meaning burning pain, was introduced by Weir Mitchell [4] in 1872. As Livingston [5] (1944) has shown, it seems clear that Mitchell used the term to describe the symptom of burning pain rather than an entire clinical syndrome. In the literature causalgia usually refers to a definite clinical entity. However, there is still a wide difference of opinion as to which signs and symptoms constitute the essential part of the syndrome.

The observations that are reported in this paper represent an attempt to determine: (*a*) some of the essential characteristics of causalgia; (*b*) the relative incidence of the signs and symptoms, and (*c*) the course of the condition when treated conservatively or by sympathectomy.

Papers have been published by those who observed and treated causalgia in the early stages overseas. Those of us who have seen peripheral nerve injuries in the later stages (in military hospitals in the United States) therefore have an opportunity to compare the inci-

Read before the American Academy of Neurological Surgery, Hot Springs, Va., Sept. 9, 1946.

1. Denmark, A.: Tr. Med.-Chir., London **4**:48, 1813.

2. Paget, J.: M. Times, March 26, 1864.

3. Mitchell, S. W.; Morehouse, G. E., and Keen, W. W.: Gunshot Wounds and Other Injuries of Nerves, Philadelphia, J. B. Lippincott Company, 1864.

4. Mitchell, S. W.: Injuries of Nerves and Their Consequences, Philadelphia, J. B. Lippincott Company, 1872.

5. Livingston, W. K.: Pain Mechanisms, New York, The Macmillan Company, 1944.

dence and manifestations of causalgia in our cases with those reported from the war theaters. Such a comparison reveals conspicuous differences. For instance, Rasmussen and Freedman[6] (1946), who were stationed in Burma, observed 100 cases of causalgia among approximately 600 cases of peripheral nerve injuries, an incidence of 16.6 per cent; whereas Mayfield and Devine[7] (1945) in this country reported an incidence of only 2 per cent in 737 cases, Speigel and Milowsky[8] (1945) 3.2 per cent in 275 cases and White[9] (1946) 2.7 per cent in 400 cases. This apparent difference in the incidence of causalgia would suggest that a considerable number of patients with the condition must have improved, at least with regard to pain, before arriving at the centers in the United States. The over-all incidence of causalgia thus may be higher than usually assumed. The analysis of our own series of peripheral nerve injuries points to this conclusion.

The war has made us causalgia conscious, and perhaps as a result the condition will be found to be commoner in civilian life than some previously suspected.

For the purposes of this paper, we have distinguished between major and minor causalgia. In using these terms we do not wish to imply that there are two forms of causalgia but, rather, that causalgia may vary in severity.

In the cases in this study regarded as instances of severe, or "major," causalgia the patients have presented essentially the clinical features described by recent authors.[10] After a wound of a large peripheral nerve, our patients have complained of an intense burning or burning and aching pain, confined chiefly to the distribution of the injured nerve or nerves in the hand or foot. The pain has been constant but subject to exacerbation on certain forms of stimulation. When persistent, it has been accompanied with changes in the tissues of the involved extremity, but the degree and nature of these changes have varied in different patients. A detailed description of the signs and symptoms is given later.

The term "minor causalgia" in this paper is applied to two types of cases: (*a*) those of patients who improved after major causalgia but continued to have moderate to mild burning pain and (*b*) those

6. Rasmussen, T. B., and Freedman, H.: J. Neurosurg. **3**:165, 1946.

7. Mayfield, F. H., and Devine, J. W.: Surg., Gynec. & Obst. **80**:361, 1945.

8. Speigel, I. J., and Milowsky, J. L.: Causalgia: Preliminary Report of Nine Cases Succesfully Treated, J. A. M. A. **127**:9 (Jan. 6) 1945.

9. Goodman, E. N.; Messinger, W. J., and White, J. C.: Ann. Surg. **124**:204, 1946.

10. (*a*) Mayfield and Devine.[7] (*b*) Spiegel and Milowsky.[8] (*c*) Goodman, Messinger and White.[9] (*d*) Rasmussen and Freedman.[6] (*e*) White, J. C.: Am. J. Surg. **72**:468. 1946.

of a small group of patients who experienced burning pain following an injury of a large peripheral nerve but gave no history of a period of severe causalgia. The time of onset, quality and distribution of the pain in all these cases of minor causalgia was the same as in the cases of major causalgia.

REVIEW OF THE LITERATURE

Pain, Trophic and Vasomotor Disturbances in Causalgia.—Mitchell, Morehouse and Keen [3] have given a classic picture of causalgia at its height. That Mitchell [4] had observed minor forms of causalgia is perhaps suggested by his statement that "its intensity varies from the most trivial burning to a state of torture which can hardly be credited."

Following Mitchell, it became customary to refer to causalgia as a syndrome and to include under the term only those cases which conformed in their severity and other manifestations to the picture presented by the patients included in Mitchell, Morehouse and Keen's original description. For instance Tinel [11] spoke of "the violent neuralgic syndrome of a particular character, for which must be reserved the name of causalgia."

Mitchell [4] was impressed with the frequent presence of glossy skin in causalgia and the belief that the two usually accompany one another has persisted. Pollock and Davis,[12] however, have shown that causalgia may exist without glossy skin and also that the reverse is true. They found 41 cases of glossy skin unassociated with causalgia in 1,020 peripheral nerve injuries. On the other hand, of 38 cases of burning pain only 8 were accompanied with glossy skin. Pollock and Davis [12] believe "It is very likely that all patients who complain of burning pain, mild or severe, with or without glossy skin, are suffering with a certain grade of causalgia."

Livingston [5] (1944) points out that in recent years the term causalgia has been used to designate a syndrome characterized by (1) burning pain, (2) extensive trophic changes of which glossy skin is the most prominent, and (3) a local rise in temperature. In his experience all of these features will be found to occur in quite variable combinations, so much so that it is a misleading and artificial distinction to select three of them and designate them as a clinical entity. Like Pollock and Davis [12] (1932), Livingston is of the opinion that it would be better perhaps if the term causalgia had been restricted as Mitchell [4] (1872) employed it, namely to describe the single symptom of burning pain.

11. Tinel, J.: Nerve Wounds, New York, William Wood & Company, 1917.
12. Pollock, L. J., and Davis, L.: Am. J. Sc. **15**:395, 1932.

Lewis [13] (1942) in remarking on the nutritional disturbances in the skin states that "These changes are no doubt, partly though not wholly, due to disuse." Doupe, Cullen and Chance [14] (1944) believe that pain is the central feature of the causalgic syndrome and that the nutritional and vascular changes, especially osteoporosis, are nonspecific.

Homans [15] (1939, 1940), Livingston [5] (1944), de Takats [16] (1945) and others have described minor forms of causalgia in civilian life.

The Incidence and Clinical Course of Causalgia.—Reports on the incidence and clinical course of causalgia in battle casualties are misleading, as only the severe, intractable cases have usually been included.

It is true as Leriche [17] (1939) said: "Sometimes far from clearing up the condition may be become steadily worse and worse; the area of distribution of the pain extending and the manifestations of vasomotor disorder continuing to spread." On the other hand, a number of observers [18] have found that in most cases the severity of the causalgic syndrome gradually abates with time. Mitchell [4] has remarked, "Many cases of burning pain last but a few weeks."

A review of Mitchell, Morehouse and Keen's [3] monograph gives some idea of the incidence of causalgia if our interpretation of their writing is correct. In discussing Paget's [2] description of the pain and tissue changes which may accompany nerve injuries they state, "That the author quoted regards these conditions as rare and of interest is the best reason why we should minutely describe them. How very common they are as results of gunshot wounds may be gathered from the fact that in 50 partial nerve lesions they existed in different degrees in 19 cases." In this description Mitchell and his associates were discussing especially the skin changes and did not state specifically that pain occurred in all cases. In the same monograph, however, they commented: "In some form pain has been an invariable attendant upon the diseased state of the skin which we have tried to describe. In the

13. Lewis, T.: Pain, New York, The Macmillan Company, 1942.

14. Doupe, J.; Cullen, C. H., and Chance, G. Q.: J. Neurol., Neurosurg. & Psychiat. **7**:33, 1944.

15. Homans, J.: Circulatory Diseases of the Extremities, New York, The Macmillan Company, 1939.

16. de Takats, G.: Causalgia States in Peace and War, J. A. M. A. **128**:699 (July 7) 1945.

17. Leriche, R.: The Surgery of Pain, translated by Archibald Young, Baltimore, Williams & Wilkins Company, 1939.

18. Mitchell.[4] Mitchell, J.: Remote Consequences of Injuries of Nerves and Their Treatment; and Examination of the Present Condition of Wounds Received 1863-1865 with Additional Illustrative Cases, Philadelphia, Lea Bros. & Company, 1895. Tinel.[11] Benisty, A.: The Clinical Forms of Nerve Lesions, edited by A. Farquhar Buzzard, Military Manuals, London, University of London Press, Ltd., 1918. Pollock and Davis.[12] Leriche.[17]

great mass of cases it has been of that peculiar burning character which we have spoken of at length . . . ." Elsewhere, writing at the same date the authors make the statement, "In fact this state of skin never existed without burning pain." From these comments it may be said with reasonable assurance that burning pain existed in approximately 19 or 38 per cent of 50 of Mitchell's patients with partial nerve lesions. Since partial nerve injuries comprise only a certain per cent (perhaps 50 to 75 per cent) of all nerve injuries, the incidence of causalgia in an unselected group of nerve injuries would be considerably lower than 38 per cent but nevertheless higher than is usually reported. These observations correspond to those to be reported in the present paper.

Pathogenesis of Causalgia.—The underlying pathologic or abnormal physiologic mechanisms which might be responsible for causalgia have been discussed at length in the recent literature and will not be reviewed in detail here. The most satisfactory explanations of the mechanism have been supplied by Weir Mitchell,[4] Livingston,[5] Doupe and associates [14] and Thomas Lewis.[13]

Mitchell [4] stated, "The immediate cause of the burning pain lies in the part where the burning is felt." To explain this he assumed "That the irritation of a nerve at the point of a wound, might give rise to changes in the circulation and nutrition of parts in its distribution and that these alterations might be themselves of a pain producing nature." Further, he remarked, "Nerve injuries may also cause pain which, owing to inexplicable reflex transfers in the centres may be felt in remote tissues outside of the region which is tributory to the wounded nerve."

Livingston [5] has postulated that the causalgic state is due to a vicious circle of activity originating in nerve impulses from a peripheral nerve, which create an abnormal state of activity in the internuncial neuron centers of the gray matter of the spinal cord. The activity in the gray matter gives rise to efferent discharges which are responsible for the abnormal muscle spasms and vasomotor and other changes in the peripheral tissues. If the process is allowed to continue, the abnormal activity in the spinal cord may become self sustaining and affect, in turn, higher centers in the central nervous system.

Doupe and associates [14] (1944) suggested that causalgia is a result of the stimulation of sensory fibers by efferent impulses in sympathetic fibers at the site of the nerve injury in some cases but possibly at a distal site near the termination of the fibers in others. The theory is based on the demonstration by physiologists [19] that interaction between

19. (*a*) Adrian, E. D.: Proc. Roy. Soc. **106**:596, 1930. (*b*) Jasper, H. H., and Monnier, A. M.: J. Cell. & Comp. Physiol. **11**:259, 1938 (*c*) Katz, B., and Schmitt, O. R.: J. Physiol. **97**:471, 1940. (*d*) Marazzi, A. S., and Lorente de Nó, R.: J. Neurophysiol. **7**:83, 1944. (*e*) Ragner, G.; Leksell, L., and Skoglund, C. R.: Brain **16**:125, 1944.

nerve fibers may occur as a result of injury. This theory is in keeping with most clinical observations on causalgia and with the fact that in the majority of cases severe causalgia can be cured by sympathectomy.[20] Doupe's interpretation of the underlying mechanism in causalgia is not incompatible with Livingston's hypothesis, but the efferent discharges over sympathetic fibers may represent only one important link in the vicious circle.

Another explanation of the underlying mechanism in causalgia is that of Lewis,[21] Gask and Ross,[22] de Takats [23] and de Takats and Miller,[24] who have expressed the belief that a release of H substance may take place into the tissues of the causalgic extremity as result of antidromic impulses (of Bayliss [25]) in the injured nerve, and that this may give rise to both pain and tissue changes.

It is quite possible that each of the three mechanisms of production of causalgia described by Livingston,[5] Doupe and associates [14] and Lewis [18] may be operating at the same time in an individual case. Actually this interpretation is not far removed from that originally postulated by Mitchell.

None of the explanations advanced in the literature regarding the underlying mechanisms responsible for causalgia conflict with the belief that the condition may improve with time and that it may manifest itself in a severe or a mild form, as regards both pain and associated changes in the tissues. Actually if Dupe's hypothesis is correct, it would seem that the symptoms and signs must rarely remain stationary and that improvement should be common. His hypothesis envisages an interaction between nerve fibers at the site of injury. An injury soon gives rise to interstitial fibrosis and other changes which in most cases would be expected to isolate and separate nerve fibers, thus preventing interaction between many of them at least.

THE STUDY

This study, as stated, is concerned with the characteristics of causalgia, its course when treated conservatively or by sympathectomy and the over-all incidence of the condition among 1,500 patients with

20. (*a*) Spurling, R. G.: Causalgia of Upper Extremity, Arch. Neurol. & Psychiat. **23**:784 (April) 1930. (*b*) Kwan, S. T.: Ann. Surg. **101**:222, 1935. (*c*) Mayfield and Devine.[7] Spiegel and Milowsky.[8] White.[10e]

21. Lewis, T.: Heart **14**:27, 1927; footnote 13.

22. Gask, C. E., and Ross, J. P.: The Surgery of the Sympathetic Nervous System, Baltimore, William Wood & Company, 1934.

23. de Takats, G.: Nature of Painful Vasodilation in Causalgic States, Arch. Neurol. & Psychiat. **50**:318 (Sept.) 1943; footnote 16.

24. de Takats, G., and Miller, D. S.: Post-Traumatic Dystrophy of Extremities, Arch. Surg. **46**:469 (April) 1943.

25. Bayliss, W. M.: J. Physiol. **26**:173, 1901.

peripheral nerve injury treated at the neurosurgical center at McCloskey General Hospital in 1945. The great majority were battle casualties that had been transferred to the United States. For the sake of analysis the patients studied were divided into three groups. Group 1 consisted of the patients with major intractable causalgia from the entire series of 1,500 cases and group 2 of patients with a history of apparent major causalgia from a series of 310 consecutive patients with nerve injury.

An attempt was made to determine what percentage of patients with peripheral nerve injuries would give a history of apparent severe causalgia even though they might be free of pain when seen. For this purpose we examined 310 consecutive patients with nerve injuries from the series of 1,500 patients with special reference to a history of burning pain, its features and course. In group 3 were included those patients, from the series of 310 in group 2, who gave a history of having had moderate to mild burning pain without any initial period of intense pain.

During the study it was observed that 15 patients with severe causalgia (grades 1 to 4) when admitted to the hospital showed sufficient improvement with time, physical therapy or sympathetic procaine block to eliminate the necessity for sympathectomy. Twelve of these patients had received one or more paravertebral injections of procaine hydrochloride, with complete temporary relief of pain in 8 and marked temporary relief in 4. In only 2 instances could one say definitely that subsequent improvement was the direct result of the procaine blocks. (Rasmussen and Freedman reported 45 cases of causalgia in which improvement followed sympathetic procaine block). The remaining 13 patients had gradual decrease of pain over a period of weeks to months, and during this interval they might be described as suffering with minor causalgia.

It was the observation of improvement in these patients that made us look for a similar phenomenon in the patients with nerve injury transferred from overseas, leading to the study reported in groups 2 and 3 in this paper.

Examination and recording has been uniform in all patients, and included in each case is a recording of the nature of the lesion, the time of onset of the pain, its quality, grade of severity, distribution, evolution and duration and a description of the objective findings. It is interesting to note that few of the routine hospital histories contain a reference to pain, except in case the patients were suffering with severe causalgia when examined. Many of the patients were surprised when they discovered that we were interested in pain which had vanished. They frequently did not mention any minor pain that

they still had unless they were questioned directly. Their minor pain was something that had lasted for a long time and that they took for granted as a natural accompaniment of their wounds.

On examining these patients we were careful, however, to avoid leading questions regarding the subjective quality of the pain. As a result, the patients included in this study spontaneously complained that the pain was burning in quality, and only patients with such pain have been included. There were many others not included who gave a history of pain of a different quality. In some of these the mechanism of production of the pain may have been similar to that operative in causalgia.

The findings in group 1 are reported first as they give a picture of the main features of classic causalgia and may be used as a yardstick in evaluating the clinical picture presented by the patients in the other two groups.

GROUP I. PATIENTS WITH MAJOR INTRACTABLE CAUSALGIA

There were 30 persons, or 2 per cent of the group of 1,500 patients with a peripheral nerve injury, who had persistent symptoms and signs of a severe or major causalgia despite conservative therapy and repeated procaine blocks of the sympathetic chain. Sympathectomy was performed on 20 of them. The other 10 who failed to improve were awaiting complete analysis and probable sympathectomy when the study was terminated.

An analysis of the main features of the cases of the 20 patients who underwent sympathectomy is presented herein, and reveals the essential similarity between these cases and the ones reported in groups II and III, in which diagnosis is based chiefly on the history. The findings were identical with those reported by many others.[26] They lend support to the thesis that pain (which, as a rule, may be temporarily relieved by sympathetic block) is the central feature of the causalgic syndrome and that the accompanying objective changes, although important, are variable and in part, at least, nonspecific.

Nature of the Lesions.—The wound was due to a shell fragment in 12 patients, a bullet in 7 and fragments of an exploding mine in 1. The arm was the site of the wound in 6, the forearm in 3, the brachial plexus in 2, the leg above the knee in 7 (in 3 of these the popliteal space was involved) and the leg below the knee in 2.

In each patient there was a partial nerve injury. In 2, however, there was pain over the distal distribution of a completely severed nerve (the ulnar in 1 and the common peroneal in the other) as well

26. Doupe, Cullen and Chance.[14] Mayfield and Devine.[7] Spiegel and Milowsky.[8] Goodman, Messinger and White.[9] Rasmussen and Freedman.[6] White.[10e]

as over the distribution of a partially injured nerve (the median in the first case and the tibial in the second). The injury involved the median nerve in 4 instances, the ulnar in 4, the radial in 1, the brachial plexus in 1 and the axillary artery as well as the brachial plexus in 1. The sciatic nerve was injured in 1 case, the posterior tibial in 2, the small sciatic in 1, the saphenous in 1, both the tibial and the peroneal in 2, and the femoral artery in addition to the sciatic nerve in 1 and the tibial in another.

There was no history of a gross wound infection in 13. In 7, however, there was drainage from the wound for two to nine months. Scarring of the soft tissues was severe in 2, moderate in 16 and slight in 2.

Symptoms and Signs.—Time of Onset of Pain: Two patients stated that burning pain was present from the moment they were wounded, and 3 said that the pain came on within a few minutes. Pain was first noted on the day following injury in 3, and within eight days in another 9. The onset was in eleven to fourteen days in 3 and on the twenty-first day in the final case. It is possible that some of these patients would have become conscious of pain at an earlier period than stated if it had not been masked by associated shock or administration of morphine.

Quality of the Pain: Burning pain of an intense character was present in 18 of the patients. Ten patients complained of aching or crushing pain as well, and in 2 of these aching was the dominant feature, although some burning was also present. Sudden stabs of pain were described in 10 and a stinging element in 3.

Severity of the Pain: The pain in the 20 cases has been graded according to Rasmussen and Freedman's classification.[6]

Grade I. These were the patients with the most severe pain. They spent most of the day huddled on their beds protecting the involved extremity. The hand or foot was frequently wrapped in a wet cloth and only the most superficial examination was allowed. The facial expression and bodily attitude were so characteristic that the diagnosis could usually be made with a passing glance.

Five of our patients presented such symptoms during the earlier weeks or months after being wounded, but 3 of them improved to grade II and 2 to grade III before sympathectomy.

Grade II. These patients likewise complained of severe pain and refused to use the part under any provocation but allowed closer examination of the painful extremity and were somewhat more active about the ward.

There were 13 patients in this group, but before sympathectomy was carried out 4 of them had improved to the status of grade III and in 2 others the severity was grade III at intervals—for instance, during the cool of the morning.

Grade III. These patients complained of moderately severe pain, used the extremity a little on occasion and allowed a fairly complete examination to be made.

There were 2 patients in this group, and neither had shown improvement.

Grade IV. The patients in this group complained of moderately severe pain but used the involved extremity somewhat more actively although still much less than their wounds would otherwise have permitted.

There were no patients in this group of 20 with such symptoms, but other patients with major causalgia presented these symptoms during the course of their improvement.

Distribution of the Pain: This has been similar to that described by other writers on major causalgia. The pain has usually been most intense in, or confined to, the hand or foot. In 5 cases, however, pain was also present in more proximal portions of the limb, and in 2 patients, 1 of whom had a lesion of the posterior femoral cutaneous nerve and the other a saphenous nerve injury, the pain did not extend into the foot. It is interesting that both patients manifested the usual symptoms of causalgia and both were cured by sympathectomy. The pain has followed quite closely the distribution of the injured nerve or nerves but the hyperalgesia and hyperesthesia to contacts has frequently been present over wider areas. It has usually been most intense in localized areas even when present over most of the distribution of a nerve in the distal portion of an extremity. In ulnar and median nerve injuries the patients have rarely complained of pain on the dorsal aspect of the hand or fingers.

Duration of the Pain: In 3 of the 20 patients undergoing sympathectomy the pain had been present for two to four months, in 17 cases for over six month, in 11 of these for ten months or over and in 5 for one year to seventeen months. It was constant in all patients but subject to exacerbation as a result of various types of stimulation

Emotional Influences: A history of an increase in the severity of the pain on emotional stress produced by perceptual stimuli, directly or indirectly, is a common feature, being present in 13 cases. For example, a paroxysmal increase in the pain was produced by a stimulus which suddenly startled, frightened or angered the patient in 11 instances. Scraping noises or rubbing a rough surface was effective in 4 patients, and in 1 the pretense of rubbing the blotter on the desk was sufficient. In 3 patients the visualization or even the thought of someone slipping or falling intensified the pain. Seven patients had never been affected by such stimulation. In 3 patients contacts with the cutaneous surface of the nonpainful hand caused pain in the opposite, causalgic, extremity.

Effect of Temperature and Moisture: Nine patients found that heat increased their pain, but 4 said it became worse with cold. In 1 patient both heat and cold intensified the pain, and 6 had noted no influence from temperature.

Seven patients, all of whom had a lesion of the median nerve, had found relief from moist applications to the painful hand, and 2 patients carried a moist cloth in both hands, although pain was felt in only one. Nine specifically stated that there had never been any beneficial effect from moisture.

Objective Signs.—The objective signs likewise varied from patient to patient. There was stiffness of the joints of the involved hand or foot in 13, and in 6 the stiffness was pronounced. Other factors than causalgia had contributed, however, to the stiffness in 3 of these. Seven patients had little or no joint stiffness.

Sweating of the painful part was increased in 10 and was excessive in 4. Sweating was absent in the distribution of the involved nerve in 4, decreased in 2 and within normal limits in 4.

The skin could be described as glossy in 2, of smooth texture in 12 and wrinkled in 6. There was a marked color change in the painful foot of 3 and some color change in 9 others. Similar skin changes, but perhaps occurring less frequently, were noted in patients with nerve injury and no history of causalgia.

A marked variability was found in the skin temperatures and results of oscillometric examinations in the different patients. The painful limb was cooler than the opposite one in 8, warmer in 5 and of approximately the same temperature in 7. Oscillometric readings in 8 cases revealed vasodilatation in 3, vasoconstriction in 3 and normal conditions in 2.

Pain on movement of the joints was present in 15, but this was severe in only 5. Hyperesthesia to light stroking and hyperalgesia to pinprick was found in 13.

There was osteoporosis of varying degree in the bones of the involved hand or foot of the 15 patients for whom roentgenograms were available. This was not infrequently observed, however, in patients with peripheral nerve injury who had no history of pain. In fact, 1 of the 20 patients just reported on had equal degrees of "spotty" osteoporosis in the two feet, although his causalgia had been confined to one extremity. This patient had an incomplete lesion of both sciatic nerves.

Examination showed slight sensory diminution and some motor weakness in the distribution of the injured nerve in 15 patients. In 1 the sensory and motor impairment was marked, and in 4 patients no deficit could be demonstrated.

Treatment.—Neurolysis was performed on the involved nerve in 8 patients without giving adequate relief from pain. There were 2 patients, already mentioned, who continued to have pain over the distribution of a nerve on which neurorrhaphy had been performed, as well as over another partially injured nerve in the same extremity.

Paravertebral injections of procaine into the sympathetic chain were carried out in all cases. Two to six injections were given to 16 patients, and on the other 4 the procedure had been carried out on eleven to twenty-five occasions. Complete temporary relief of pain for an hour to a number of days occurred in 17. Partial temporary relief only was experienced by 3. None of these 20 patients had adequate permanent relief and hence sympathectomy was performed.

Preganglionic sympathetic denervation of the arm was performed in 11 cases. This was done according to Smithwick's [27] technic in 6 instances. In 5 additional cases the same technic was used, but the fourth rib was removed instead of the third. In these cases the anterior and posterior roots of the third and fourth intercostal nerves as well as their gray and white rami were sectioned, instead of the second and third roots, as is usual, and the sympathetic chain was divided below the fourth instead of the third thoracic ganglion. Four of the 5 patients were cured of their pain, despite the fact that the first and second thoracic ganglions as well as the first and second intercostal nerves, remained intact. Sweating tests on the 5 patients demonstrated apparent complete sympathetic denervation of the upper extremity.

Eight of the 11 patients were free of their pain and hyperesthesia after thoracic sympathectomy and remained so, at least during the two to six months that they were under observation. One patient obtained no relief. Preoperatively this patient had experienced only about 50 per cent relief of his pain after each of three paravertebral blocks. One patient, although much improved, continued to complain of some burning over the median nerve distribution in the hand. The final patient was free of pain except for slight aching over an area ½ inch (1 cm.) in diameter on the anterior lateral aspect of the index finger and a similar area on the anterior surface of the thumb. All burning pain had disappeared and the joints could be moved freely without pain in this patient. The last 2 patients had preoperatively been completely relieved temporarily by sympathetic block.

Lumbar sympathectomy for causalgia in a lower extremity was performed on 9 patients, the second, third and fourth lumbar ganglions with the intervening chain being removed in each case, through a muscle-

27. White, J. C., and Smithwick, R.: The Autonomic Nervous System: Anatomy, Physiology, and Surgical Application, ed. 2, New York, The Macmillan Company, 1941.

splitting retroperitoneal approach. Seven patients obtained complete relief of their pain and hyperesthesia following operation. One had complete relief of burning in the sole, but persistent burning remained in the toes and dorsum, and this was aggravated by walking. The other patient with pain in the sole obtained no relief except for three days after operation. Subsequently a neurorrhaphy of the tibial nerve at the site of his wound at the ankle abolished the pain. Both of these patients preoperatively had had temporary disappearance of pain following paravertebral injections of procaine. Twenty-four procaine blocks had been performed on the last patient, with complete temporary relief following 15 of these, varying from two hours to four days.

In the patients in whom pain was relieved by sympathectomy there was a remarkably rapid return of the emotional state to normal levels if there had been emotional disturbances prior to the operation.

In our study we have had no experience with general anesthesia or anesthesia combined with psychotherapy as methods of treatment in causalgia. Zikeyev [28] reported that he was able to cure 34 of 35 patients with severe causalgia by placing them under general anesthesia, followed by narcosis, for one hundred and twenty to one hundred and sixty-eight hours. Lidz and Payne [29] and Pool have each reported a case in which the patient was cured by psychotherapy while under the influence of pentothal sodium® administered intravenously.

GROUP II. PATIENTS WITH A HISTORY OF APPARENT MAJOR CAUSALGIA

Examination of 310 consecutive patients (from the series of 1,500) with injuries of one or more major peripheral nerves revealed that 61 of these, or 19.6 per cent, had suffered for varying periods with an intense burning type of pain. The time of onset, quality, severity, characteristics and distribution of the pain in these cases appeared indistinguishable from those described in group I as occurring in cases of intractable causalgia that have been cured by sympathectomy.

The 310 patients were all soldiers who had received an injury of a peripheral nerve. The injury was due to a shell fragment, grenade fragment or bullet wound in 296 instances. In 4 it was the result of a stretch injury, and in 10 it was due to a laceration from glass or other sharp object.

Nature of the Lesions.—Of the 61 patients with a history of intense burning pain, 59 were battle casualties. Shell fragments, grenade fragments or bullets were responsible for the wounds in 59 patients. One patient received a stretch injury of the brachial plexus and another a laceration from glass. The upper extremity was involved in 30

28. Zikeyev, V. V.: Am. Rev. Soviet Med. **4**:16, 1946.

29. Lidz, T., and Payne, R. L., Jr.: Causalgia: Report of Recovery Following Relief of Emotional Stress, Arch. Neurol. & Psychiat. **53**:22 (March) 1945.

patients and the lower in 31. The injury involved the median nerve in 8, the ulnar in 6, the radial in 1 and the brachial plexus in 5. There was a combined lesion of the ulnar and median nerves in 7 patients, and the brachial artery was injured in addition to a major nerve in 3. There were 18 lesions of the sciatic nerve, 6 of the tibial or posterior tibial, 3 of the peroneal and 4 combined lesions of the tibial and peroneal nerves. In 12 of the 61 patients the nerve lesion was clinically complete or so nearly complete that neurorrhaphy was performed. Whether a few fibers were still intact in most of these patients we are unable to say definitely. In the remaining patients the nerve lesions were partial.

Symptoms and Signs.—Time of Onset of Pain: Eighteen of the patients stated that the burning pain was present from the moment of impact of the missile. In 12 the onset was on the day of injury; in 10 it was first noted within two to four days, and in 17, within six to fourteen days. In only 4 cases was the onset delayed longer than three weeks. These observations are in keeping with those of Mayfield and Devine,[7] Rasmussen and Freedman [6] and others and also with our own on a considerable number of patients with major causalgia who were cured by sympathectomy.

The Quality of the Pain: During the first weeks or months after receipt of the wound, an intense burning was the predominant element of the pain described by 53 of the 61 patients. Twenty-one also complained of aching of varying severity, and in 8 patients this represented the chief complaint, although burning pain was also prominent.

The burning element was described in various ways, as "like a blaze of fire," "like some one was pouring boiling water on the top of my foot and holding a cigaret lighter under my big toe," "burning like on fire," "burning, just burning," "like my hand was pressed against a hot stove," "sharp burning," "like a hot foot."

Severity of the Pain: The severity of the pain is difficult to grade with accuracy, since in most cases it had reached a marked degree of improvement at the time of examination. From the history, however, we believe the pain was sufficiently severe during its earlier stages to place all these 61 patients in the class of major causalgia (grades I to III) if intensity alone could be used as a criterion.

In over 85 per cent of the patients the burning was constant in character, but subject to sudden fluctuations in most, during that period of weeks or months when it was at its height. Although most of the patients could recall vividly paroxysmal exacerbations of their pain, many of them could not remember accurately whether emotional stress had been responsible. We cannot, therefore, give a reliable estimate of the effect of emotion on the pain experienced by this group of patients.

Distribution of the Pain: The distribution of the pain in these 61 patients appeared strikingly similar to that seen in patients with causalgia cured by sympathectomy. The burning was most intense in the hand or foot in all, although some pain, especially of an aching character, was present in the arm or leg in at least 6 patients.

In most patients the pain was largely confined to the distribution of the involved nerve or nerves, but was frequently more intense in some areas than in others. In 2 of 7 patients with combined ulnar and median nerve lesions, however, the pain was described as involving the whole hand. This was true also in 2 of 5 with brachial plexus injuries and in all 3 patients in whom there was severance of the brachial artery in addition to an injury of a major nerve. The whole foot was described as the site of burning in 6 of 18 with sciatic nerve lesions.

With time as the pain gradually diminished the tendency for the burning to involve a restricted area commonly became more pronounced. In some patients the burning in the hand gradually disappeared except over the terminal phalanx of the thumb and index finger. In the foot the burning not infrequently became restricted to the region of the instep, around the heel or under the plantar surface of the toes. These localized pains frequently became intermittent as the pain improved.

Evolution and Duration of the Pain: In 15 patients the pain had gradually disappeared spontaneously; in 6 it had been cured by neurorrhaphy; in 2 it was cured by sympathectomy performed within four weeks after the initial wound and in 1 it had disappeared after a single procaine sympathetic block. In 24 patients the severe pain, although it did not clear up completely, gradually and spontaneously became minor in form with time and physical therapy as the only treatment. Eight patients improved after operations on the peripheral nerve involved (neurorrhaphy in 4, neurorrhaphy of one nerve in 1 and neurolysis of another in 4), but 2 showed no improvement with neurorrhaphy.

As to the duration of severe burning pain, this had, without treatment other than physical therapy, either disappeared completely or become relatively mild in form (grade V—sufficient pain to cause some interference with work—to VI—pain not severe enough to interfere with work) within two and a half months in 23 of the 61 patients and within three to six months in an additional 12. On the other hand, severe burning, with its consequent fixation of the part and possible accompanying trophic and vasomotor disturbances, had persisted for over three months in 31 and over six months in 10 (an incidence of 3.2 per cent in 310 patients with peripheral nerve injuries). Five patients, or 1.6 per cent, continued to have severe pain when examined three months to one year after the onset.

In addition, it should be noted that 32 of the 61 patients, although improved to a status where sympathectomy no longer seemed indicated, were still suffering with some pain at the time of the present study. In 26 this had been present for over six months, in 13 over ten months and in 6 over seventeen months.

Of the 32 patients who continued to have pain at the time of this study, 20 described it as mild in severity (grade VI) and 12 as moderate (grade V). In 28 a burning element was still present, and in 10 some hyperalgesia persisted over the distal distribution of the involved nerve. In 4, all burning had subsided but aching persisted. Twelve patients stated that the pain was now definitely intermittent.

Objective Findings.—Data on the objective findings have been recorded on only 30 of the patients. Of these, 21, or 70 per cent, showed some stiffness of the joints and in 3 it was marked. Sweating was increased in 12, diminished in 8 and within normal limits in 10. Changes in the color of the hand or foot were present in 22. This was slight in 16. In most of these patients the skin was somewhat pinker than normal and had a slightly mottled appearance. The skin was a dusky cyanotic color in 4, and there was a brawny induration in 2. Definite glossy skin was present in only 3 of the 30 patients, although the skin was smoother than normal in at least 10.

On the basis of the material available, we believe that those patients who had suffered pain for a period of months showed more irreversible tissue changes than a comparable group of patients with peripheral nerve injuries but without a history of prolonged pain. A more detailed study of this aspect of the problem is worth while.

GROUP III. PATIENTS WITH A HISTORY OF APPARENT MINOR CAUSALGIA

Among the 310 patients with an injury of a major peripheral nerve reported in group II of this paper, there were 26, or 8.3 per cent, who complained of a moderate to mild burning pain in the hand or foot which commenced when they were wounded or shortly thereafter and in many cases persisted for months. There was no history of a period of intense pain in these patients as in the 61 previously described in group II, and there were no marked trophic or vasomotor changes attributable to a specific syndrome. We draw attention to these patients because of the marked resemblance they bear to patients who have shown improvement following a major causalgia but who are still suffering pain. The quality of the pain and its time of onset and distribution are strikingly similar in the two types of case. We have been able to investigate the effect of paravertebral block in only 3 of these patients, and in these there was complete temporary disappearance of the burning pain following the procedure. The supposition is that these

cases at least, and probably the others as well, represent a minor form of causalgia.

All the patients were battle casualties, with 3 exceptions. Shell fragments, grenade fragments or bullets were responsible in 23 cases. Two patients received a laceration from glass, and 1 had a stretch injury. The upper extremity was the site of the wound in 14 and the lower in 12. There was an injury of the median nerve in 4 patients, the ulnar in 4, the radial in 2, the brachial plexus in 1, and a combined lesion of the ulnar and median nerves in 3. The sciatic nerve was involved in 5 patients, the tibial or posterior tibial in 5, the peroneal in 1 and both the peroneal and the tibial in 1.

Time of Onset of the Pain: This was said to be immediate in 8, within a few hours in 2, on the day of wounding in 2, two to four days in seven, six to fourteen days in five and in one month in 2.

Quality of the Pain: The pain was of definite burning character in all patients, but aching was also present in 4, drawing pain in 1 and stinging in 4. Eighteen of the patients described the pain as moderate (grade V), and 8 said that it was mild (grade VI).

Distribution of the Pain: The distribution of the pain was essentially similar to that described in the previous group. It was confined to the hand or foot in 21 patients, but extended to the wrist in 1, the forearm in 3, and the lateral part of the calf in 1. The whole hand was the site of pain in 1, a brachial plexus injury. All the fingers were involved in 2, and all the finger tips in 1, a combined lesion of the ulnar and median nerves. In the majority of patients the burning was most noticeable in relatively discrete areas in the distribution of the involved nerve in the hand or foot. In the hand the pain was largely, if not entirely, confined to the palmar aspects or sides of the fingers, except in patients with radial nerve lesions, of whom there were 2.

Evolution and Duration of Pain: In 5 patients the pain disappeared spontaneously, in 3 following neurorrhaphy and in 1 after neurolysis. Seventeen of the patients continued to have pain when this study was undertaken. Ten said it had remained the same since its onset and was constant or almost so, but in 7 there had been some improvement and the pain was definitely intermittent. In 7 it had been present three to six months and in 9 over seven months.

SUMMARY AND COMMENT

Mitchell, Morehouse and Keen,[8] in their classic description of causalgia, emphasized the severest forms of the condition and hence gave a picture of the syndrome at its height. As mentioned in the discussion of the literature, it has since become customary to restrict use of the term causalgia to those intractable cases which conformed in the severity of the pain and trophic, vasomotor and other manifestations

to the severe forms included in the classic description. On the basis of the present study there are at least two objections to this definition, or classification, of causalgia.

The first objection is that such a definition excludes many less severe, but nevertheless genuine, examples of causalgia. For instance, according to this definition, the term causalgia could not be used to describe the moderate to mild burning pain which many patients experience after an initial episode of severe, or major, causalgia. The definition also excludes the pain of patients who present what we regard as symptoms of minor causalgia, without ever having suffered from major causalgia. In the present study many examples of both these types of minor causalgia have been observed to follow wounds of the peripheral nerves.

The second objection to this definition of causalgia is that it includes certain nonessential criteria. Pain is the central feature of causalgia. Reflex vasomotor and trophic changes frequently are present in severe forms, but, as reported by others, they were very variable in even our patients with the severest manifestations and were in large part nonspecific. Such changes are, therefore, not an essential part of the syndrome, and their absence, especially in mild forms, does not preclude the diagnosis of causalgia.

It is our opinion that Mitchell [4] did not wish to imply that in all cases causalgia is severe, nor that in all cases is there progress to a chronic stage in which trophic and vasomotor changes in the involved extremity become pronounced. In Mitchell's experience in most cases there was improvement with time. For instance, he remarked: "The curative treatment was simple. In light cases two or three blisters have answered; in others, ten or twelve have been needed, and, in very rare examples, this and every method failed us, although such was never the case in any instance which was treated early, only the oldest cases being thus obstinate."

Only 2 per cent of our 1,500 patients with peripheral nerve injuries (group I in the body of the paper) continued to suffer with relatively intractable, severe or major causalgia, despite conservative therapy and repeated procaine sympathetic blocks and required sympathectomy. This is in keeping with the 2 to 3.7 per cent reported by other observers as descriptive of the general incidence of causalgia in the recent war.

In our study of causalgia it soon became evident that the total incidence of the condition is much higher than the cited figures indicate. We observed that 15 patients with major, or severe, causalgia showed such marked improvement while under observation that sympathectomy was no longer indicated. The patients referred to were relieved temporarily by diagnostic sympathetic procaine block, but lasting improvement occurred only gradually over a period of months in 13 and in these

could not be attributed to the block. These facts suggested the probability that other patients with causalgia from war wounds had also improved before arriving in the United States and were hence being excluded from the recent reports on the incidence of causalgia, which usually have included only the severe cases.

To test this assumption that cases of causalgia were being overlooked because of improvement in symptoms, we examined 310 consecutive patients (group II in the body of the paper) from among our 1,500 patients with nerve injury. Sixty-one of them, or 19.6 per cent, gave a history of what we believe in most cases to have been a major causalgia. All 61 had suffered for weeks to months with a severe burning pain which in its time of onset, quality, severity, characteristics and distribution was indistinguishable from that seen in patients with causalgia cured by sympathectomy. Fifty-six of the 61 were improved or free of pain at the time of admission to the hospital. Gradual spontaneous improvement in the intense burning pain had occurred within two and one-half months from the time of its onset in 37.7 per cent and within six months in an additional 19.6 per cent. Severe burning pain had been present, however, for over three months in 31 patients, or 50.8 per cent of the 61 patients, and for over six months in 10, or 16.3 per cent. Fifty-two and four-tenths per cent continued to have the symptoms of a mild, or minor, causalgia when examined.

The course of the syndrome in most of these 61 cases, in which the diagnosis of major causalgia was based largely on the history, has appeared identical to that observed in cases of major causalgia with gradual improvement while under direct observation. In all these cases the condition was characterized by constant pain, usually of a burning or burning and aching character, confined chiefly to the distribution of the injured nerve in the distal part of the involved extremity. The pain at first was severe, and, in most cases, subject to sudden exacerbations on certain types of stimulation, which did not necessarily have to be applied to the injured limb. Movement or contacts with the painful part usually precipitated a paroxysm of pain. Trophic and vasomotor changes frequently took place in the extremity which the patient held immobile for fear of intensifying the pain. As time passed, however, the intensity of the syndrome usually abated and the burning became less severe and, although following the same distribution, was more restricted to certain areas in the hand or foot. Sudden paroxysms in the pain became progressively harder to provoke, movement of the hand or foot was less painful, and trophic and vasomotor changes diminished as movement became possible. During this phase of the condition the patient was still suffering with causalgia, but it no longer had the intensity present in the patients included in the classi-

cal description of the syndrome given by Mitchell and associates.[3] It had, in fact become what could be called a minor causalgia.

In some patients (8.3 per cent of 310 with nerve injuries, group III in the body of the paper) there was no history of a major causalgia but symptoms of a minor causalgia essentially similar to those just mentioned appeared shortly after injury and persisted for months.

These observations provide strong evidence that there are many degrees of causalgia. Needless to say, we do not wish to quarrel with any author who prefers to reserve the term causalgia for the severe form of the condition, in which the clinical syndrome is fully established. It is only in the severe cases that one witnesses the complete syndrome. As the patient improves the range of stimuli which precipitate a paroxysm becomes increasingly narrow, and reflex, vasomotor and trophic changes diminish. Finally a point is reached at which most clinicians are reluctant to employ the term causalgia.

However, regardless of terminology, the same abnormal mechanisms which produce the signs and symptoms of major causalgia continue to operate (if only with less momentum) as the severity of the condition gradually abates, for in such cases the pain frequently remains essentially the same in quality and distribution, although its intensity diminishes and paroxysms may no longer be provoked by stimuli which are not directly applied to the affected limb.

The same mechanisms appear to be functioning in those of our patients who have shown relatively mild or minor clinical manifestations of causalgia from the onset and in whom the full syndrome has never developed.

CONCLUSIONS

1. Pain is the central feature of causalgia. Vasomotor and trophic changes are variable and for the most part nonspecific. They are not an essential part of the syndrome.

2. Severe, or major, causalgia resulting from war wounds of large peripheral nerves is commoner than has usually been reported.

3. The majority of patients with major causalgia show gradual spontaneous improvement.

4. The classic syndrome of causalgia is seen in its complete form only during the period that the condition is at its height.

5. As improvement occurs in a case of causalgia, all gradations of intensity in the condition may be seen.

6. During the period of improvement in a case of severe causalgia, the condition may be described as one of minor causalgia.

7. Some patients present the picture of a minor causalgia without ever passing through a phase of severe or major causalgia.

8. Realization that causalgia may exist in a mild, or minor, form is important, as the condition is probably more common in civilian life than generally recognized.

9. The same abnormal mechanisms are apparently operative in all cases of causalgia, regardless of intensity.

10. Although gradual improvement is usual in a case of causalgia, this may not take place for months or even years in some cases. In the interval progressive and irreversible changes may occur in the tissues and perhaps even in the emotional stability of the patient. In addition, the patient may be left with a disagreeable minor causalgia. Adequate treatment should, therefore, be instituted early.

11. An injured nerve should be appropriately treated on its own merits. Coexisting causalgia may thereby be relieved, especially if neurorrhaphy is the procedure of choice. The individual case of causalgia should be evaluated with respect to its duration, severity and response to the conservative expedients of physical therapy and sympathetic block. These measures failing, sympathectomy should be done in cases of severe causalgia if sympathetic block has given temporary relief. In such circumstances sympathectomy has been curative in the majority of patients, at least during the period of their follow-up.

STUDIES ON THE OCCIPITAL LOBE

1. Significance of Small Areas of Preserved Central Vision

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THE localization of intracranial mass lesions by means of hemianopsic defects has long been one of the standard methods used in neurologic diagnosis. The significance of macular "sparing," however, is still cause for controversy. Its presence or absence often serves to localize a lesion anywhere from the optic chiasm back to the calcarine cortex. The term macular "sparing" is a figurative one and has been used to include sparing of central vision varying from 2 to 10 degrees.[1] Thus, homonymous hemianopsia without sparing was believed to point to a lesion of the optic tract, whereas one with so-called macular sparing tended to indicate a lesion of the occipital lobe.[2] Homonymous quadrantic cuts are considered significant of lesions between the lateral geniculate body and the calcarine cortex, involving the optic radiation.[3] Since clinical evidence has shown that macular "sparing" may occur in the case of a large lesion of the occipital lobe, numerous hypotheses have been advanced to explain this phenomenon. The anatomy of the optic pathways and its fiber relations are gradually being worked out. It remains to determine the exact area of termination of the macular fibers and to decide the present status of the theory of bilateral macular representation.

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2. Kestenbaum, A.: Clinical Methods of Neuro-Ophthalmologic Examination, New York, Grune & Stratton, Inc., 1946, p. 115.

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It is known that in man the primary neurons of the visual system originate in the eighth,[4] or so-called ganglion, layer of the retina and course backward as the optic nerve. Approximately half the fibers of each optic nerve course in the chiasm to the opposite side of the brain. These fibers come from the nasal half of the retina. They continue back, with the opposite temporal fibers as the optic tract, to the lateral geniculate body.[5] Here they form synapses with neurons of the second order. These neurons of the second order pass backward as the geniculocalcarine tract, or optic radiation, and terminate in the area striata. The inferior lateral portion of the radiation bends forward and swings closely around the sides of the temporal horn of the lateral ventricle, as pointed out by Meyer and Archambault.[6] It then passes posteriorly to terminate in the calcarine cortex. The superior medial portion courses backward more directly along the body of the lateral ventricle as it descends, before terminating in the upper calcarine lip. The lower retinal quadrant is represented in the temporal detour, and the superior retinal quadrant, in the superior medial limb of the geniculocalcarine tract. In man, the majority of the macular fibers are believed to terminate in the occipital poles [7] or in the posterior portion of the calcarine cortex. Nevertheless, there are a number of cases on record [8] in which the macula was spared although all but a remnant of the anterior portion of the area striata had been destroyed. Numerous methods have been used in an attempt to explain homonymous hemianopsia with macular sparing in cases of lesions of the occipital lobe. These include anatomic

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dissection, animal experimentation, degeneration studies and perimetric studies following occipital lobectomy. The last method has been the most fruitful, with increased accuracy in perimetry and fixation.

The present studies were undertaken with the idea of carrying out accurate studies of the central fields, using the same conditions and personnel. Six cases of occipital lobectomy on the left side are presented, with studies carried out from two to fourteen years after operation. For all the patients central visual fields were mapped out repeatedly, using the Lloyd stereocampimeter and the Bjerrum tangent screen. Electroencephalographic studies were likewise performed on each patient. A report on this part of the work will follow. In carrying out the present study, one of us (G. A.) either personally conducted or was present during each examination of the visual fields. None of the studies of the visual fields was done by a technician. Identical lighting conditions were employed in each instance. The subjects were carefully observed for any sign of slight visual shift. Whenever there was doubt as to accuracy of fixation mapping of the entire field was repeated. All subjects were accustomed to the Bjerrum tangent screen and had had previous experience with it. The line of ablation of the occipital lobe was located as exactly as possible from the pathologic specimen and from the operative notes. Our landmarks were determined by the amount of ventricle exposed and the line of excision in relation to the parieto-occipital fissure.

REPORT OF CASES

CASE 1.—P. K., a miner aged 40, was referred by Dr. G. G. Reese from Shamokin, Pa. The patient was admitted to the Hospital of the University of Pennsylvania to Dr. Charles H. Frazier's service on Sept. 16, 1932. The patient complained of headache and progressive loss of vision, with onset of symptoms approximately three weeks prior to admission. He had been a coal miner for twenty years and had continued working at his usual job until the day before his admission to the hospital. The following significant signs were elicited on physical examination: slight mental dulling; indefinite past pointing and definite adiadokokinesis with the left hand; gross right homonymous hemianopsia; bilateral papilledema of 6 D.; hyperactive tendon reflexes bilaterally, and an area of hyperpathia in the left temporal region. The results of blood studies, urinalysis and serologic tests were essentially normal. Perimetric examination revealed right homonymous hemianopsia with central sparing. Roentgenograms of the skull showed a large area of calcification in the left occipital region. The pineal body was displaced to the right. On September 23 a left occipital osteoplastic craniotomy was carried out by Dr. Francis C. Grant, with the use of tribromoethanol solution U. S. P. and local anesthesia. A yellowish area of discoloration was disclosed over the left occipital pole. The tumor was partly excised and the left occipital lobe amputated. The line of excision was carried down anterior to the parieto-occipital fissure through the posterior horn of the ventricle, exposing the choroid plexus. The patient was discharged from the hospital asymptomatic, except for the right homonymous hemianopsia, on November 8. The pathologic diagnosis was hemangioma.

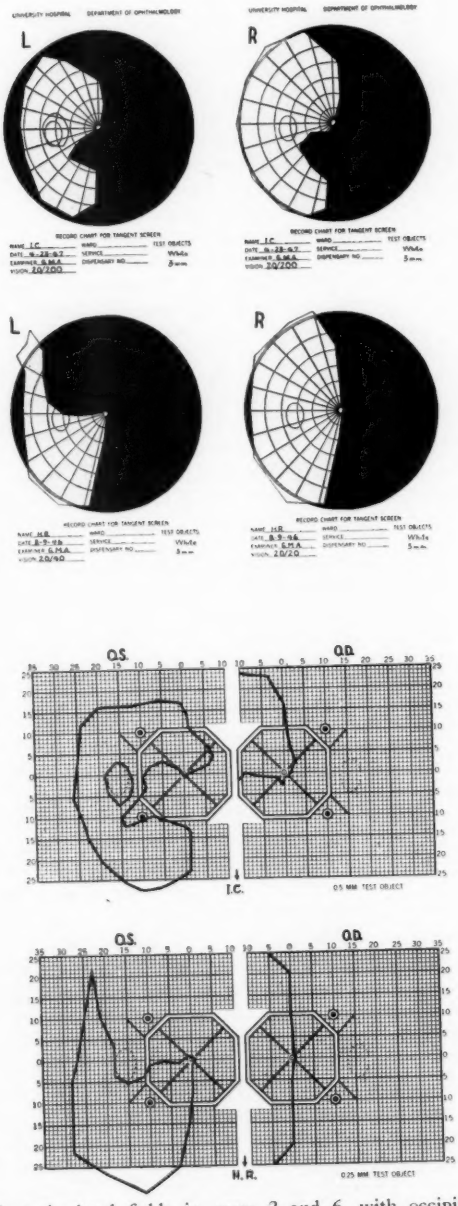

Fig. 1.—Central visual fields in cases 2 and 6, with occipital lobectomy on the left.

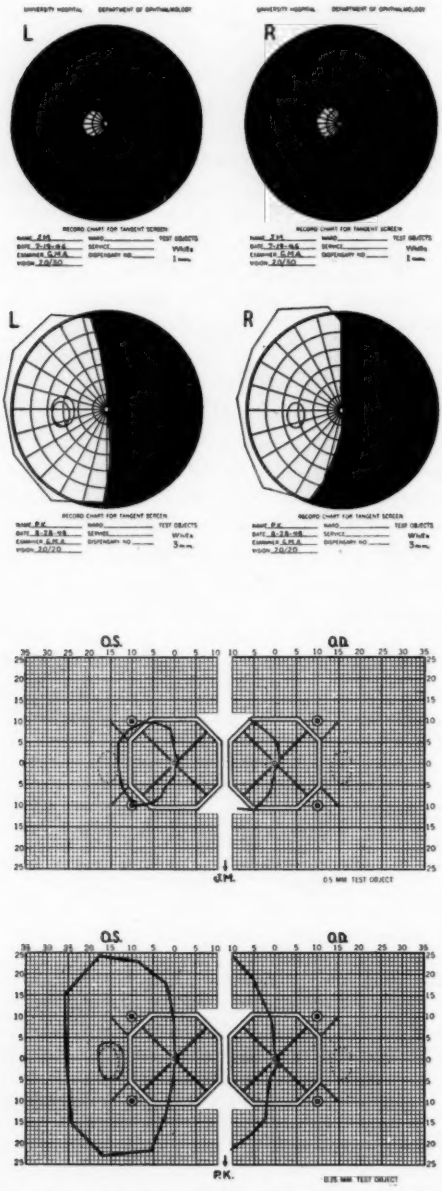

Fig. 2.—Central visual fields in cases 1 and 3, with occipital lobectomy on the left.

Examinations of the central visual fields, carried out on May 29 and Aug. 28, 1946, revealed preservation of central vision up to 0.75 degree on repeated trials with the Lloyd stereocampimeter and the Bjerrum tangent screen (fig. 1). Different-sized test objects were used, and the patient was closely observed for any shifting of vision. The results of the field studies are listed in table 1.

CASE 2.—H. R., a clerk aged 22, was referred by Dr. Edward Rubin of Philadelphia. The patient was admitted to the neurologic service of the Hospital of the University of Pennsylvania on April 11, 1940. His symptoms appeared in 1930, when he first noted epileptiform seizures of the right side occurring approximately once a day. There was no associated aura, and the attacks gradually decreased in frequency until 1934, when they ceased entirely. From 1934 to 1940 the patient remained asymptomatic. In January 1940 he noted a dull occipital headache, which gradually increased in severity. He was also conscious of gradual blurring of vision and occasional nausea and vomiting. The following pertinent signs were elicited on physical examination: bilateral papilledema of 6 D. and slight weakness of the right side of the face, increased tendon reflexes and absence of abdominal reflexes on the right side, moderate nuchal rigidity and mental dulness. Ventriculographic study on April 23 revealed a large tumor of the left occipital lobe, and the patient was discharged, to be readmitted later for operation. Roentgenograms at this time revealed a shift of the pineal body to the right. Peripheral fields at the time revealed a suggestive right homonymous hemianopsia. On June 10 a left parieto-occipital osteoplastic craniotomy was carried out by Dr. Francis C. Grant. A large glioma lying against the falx was removed, and the left occipital lobe was amputated. The line of excision passed anterior to the parieto-occipital fissure and through the poserior horn of the ventricle. The patient made an uneventful recovery and was discharged on June 23. There were slight residual weakness of the right hand and minimal mixed aphasia. Pathologic examination of the tumor revealed a neuroblastoma.

Examinations of the central visual fields were carried out on Aug. 9, 1946 for the purposes of this study. Tests with the Bjerrum tangent screen revealed preservation of central vision varying from 0.5 to 0.75 degree bilaterally. Plotting was done at a distance of 1 meter with different-sized white test objects. Tests with the Lloyd stereocampimeter, using a 0.25 degree test object (1.5 mm.), showed preservation of central vision from 0.25 to 0.5 degree (table 1).

CASE 3.—J. M., a carpenter aged 35, referred by Dr. George Hill of Summit Hill, Pa., was admitted to the neurosurgical service of the Hospital of the University of Pennsylvania on May 24, 1943. The patient complained of right-sided headache and difficulty in judging distances, the symptoms appearing four months prior to his admission. Owing to his poor vision, he became involved in two automobile accidents while driving to work. The following significant signs were elicited on physical examination: slight right hemiparesis, Babinski and Oppenheim reflexes on the right, gross right homonymous hemianopsia and papilledema of 1 D. bilaterally. The results of blood studies, urinalysis and serologic tests were normal. Roentgenograms of the skull likewise showed a normal condition. A ventriculogram taken on May 31 revealed a large tumor of the left occipital lobe. A left parieto-occipital osteoplastic craniotomy was then carried out by Dr. Francis C. Grant, with amputation of the left occipital lobe and what seemed to be complete removal of the tumor. The incision began anterior to the parieto-occipital fissure and was carried down through the ventricle, including 1 cm. of the posterior horn in the excised lobe. Pathologic examination of the tumor revealed a fibrillary astrocytoma.

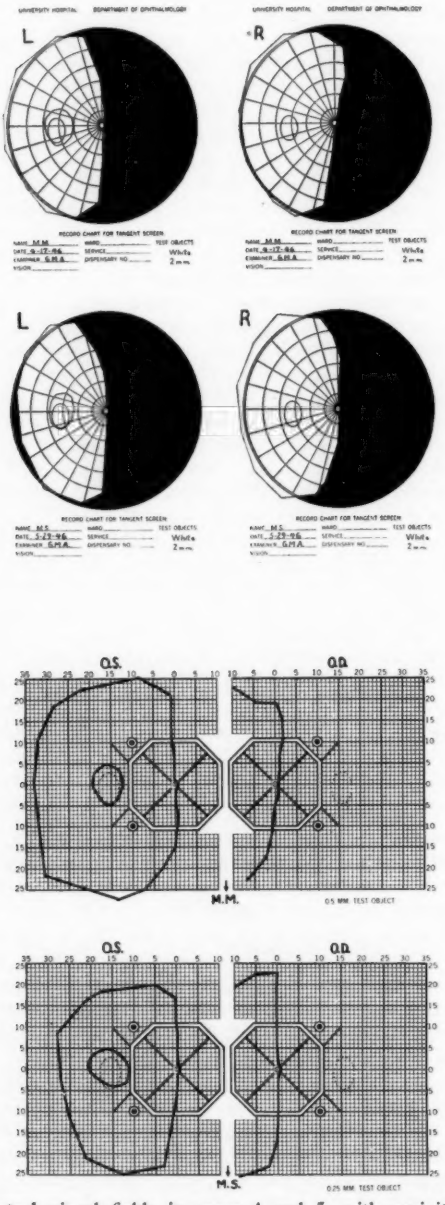

Fig. 3.—Central visual fields in cases 4 and 5, with occipital lobectomy on the left.

TABLE 1.—*Preoperative and Postoperative Studies in 6 Cases with Occipital Lobectomy on the Left*

Case No., Initials, Sex, Age, Operative Date	Visual Acuity		Preoperative Visual Fields	Amount of Preserved Central Vision in Postoperative Visual Fields		Amount of Excision	Type of Tumor	Meridional Extension into Blind Field
	Pre-operative	Post-operative		Tangent Screen	Stereo-campimeter			
1 P. K. M 40 9/23/32	O.D.:6/ 6, no cor. O.S.:6/12, no cor. (9/19/32)	O.D.:6/ 6, no cor. O.S.:6/12, no cor. (8/28/46)	Right homonymous hemianopsia with sparing approximately 10° bilaterally (Ferree-Rand perimeter)	1:O.D. 0.75° O.S. 0.75° 2:O.D. 0.5° O.S. 0.75° 1.5/1,000 and 3/1,000	1:O.D. 0.75° O.S. 0.75° 2:O.D. 0.75° O.S. 0.75°	1. Parieto-occipital fissure (anterior) 2. Choroid exposure	Hemiangioma of occipital pole	1:O.D. & O.S. 0-1° 3/1,000 white; tangent screen
2 H. R. M 20 6/10/40	O.D.:6/ 9, no cor. O.S.:6/ 9, no cor. (6/6/40)	O.D.:6/ 6, no cor. O.S.:6/12, no cor. (8/9/46)	Contracted fields with suggestive right homonymous hemianopsia peripheral and central fields	1:O.D. 0.75° O.S. 0.75° 2:O.D. 0.75° O.S. 0.75° 3/1,000 and 5/1,000 white	1:O.D. 0.5° O.S. 0.25° 2:O.D. 0.5° O.S. 0.25°	1. Slightly anterior to parieto-occipital fissure 2. Ventricle opened, amount unknown	Neuroblastoma of occipital lobe	1:O.S. 0° O.D. 1° 3/1,000 white; tangent screen
3 J. M. M 35 5/31/43	O.D.:6/ 6, no cor. O.S.:6/ 6, no cor. (5/24/43)	O.D.:6/ 9, no cor. O.S.:6/ 9, no cor. (7/19/46)	Partial right homonymous hemianopsia peripheral and central fields	1:O.D. 0.25° O.S. 0.25° 2:O.D. 0.5° O.S. 0.5° 1/1,000 and 1.5/1,000 white	1:O.D. 1.0° O.S. 0.5° 2:O.D. 0.75° O.S. 0.5°	1. Parieto-occipital fissure (anterior) 2. Ventricle opened, 1 cm.	Fibrillary astrocytoma of occipital pole	No extension in either eye
4 M. S. F 22 5/11/44	O.D.:6/ 9, no cor. O.S.:6/18, no cor. (5/7/44)	O.D.:6/15, no cor. O.S.:6/12, no cor. (7/16/46)	Partial right homonymous hemianopsia peripheral and central fields	1:O.D. 0.75° O.S. 0.75° 2:O.D. 0.75° O.S. 0.5° 1.5, 2 and 3/1,000 white	1:O.D. 0.25° O.S. 0.25° 2:O.D. 0.5° O.S. 0.25°	1. Approximately through parieto-occipital fissure 2. Ventricle opened, 0.75 cm.	Metastatic rhabdomyosarcoma of occipital pole	1:O.D. 2° O.S. 0° 2:O.D. 3° O.S. 1°
5 M. M. F 34 12/22/44	O.D.:6/22, no cor. O.S.:6/22, no cor. (9/19/44)	O.D.:6/12, no cor. O.S.:6/12, no cor. (7/15/46)	Partial right homonymous hemianopsia peripheral fields	1:O.D. 1.5° O.S. 1.0° 2:O.D. 0.5° O.S. 0.75° 3:O.D. 1.0° O.S. 1.0° 1.5, 2 and 3/1,000 white	1:O.D. 1.0° O.S. 1.0° 2:O.D. 1.0° O.S. 0.5°	1. Approximately through parieto-occipital fissure 2. Ventricle not visualized	Meningioma arising from anterior tentorium	2:O.D. 2° O.S. 0.5° 3:O.D. 3° O.S. 1° Tangent screen
6 I. C. F 53 9/12/45	O.D.:6/60, no cor. O.S.:6/60, no cor. (7/17/46)	O.D.:6/60, no cor. O.S.:6/60, no cor. (7/17/46)	Right homonymous hemianopsia with sparing of 5° peripheral fields	1:O.D. 1.0° O.S. 1.0° 2/1,000 and 3/1,000 white	1:O.D. 1.5° O.S. 1.25°	1. Approximately through parieto-occipital fissure; ventricle not visualized	Meningioma arising from tentorium	1:O.S. 6.5° O.D. 2.5° Stereocampimeter

Determinations of the central visual fields were carried out on July 19, 1946. Tests with Bjerrum tangent screen showed preservation of central vision up to 0.5 degree bilaterally. Different-sized white test objects were used at a distance of 1 meter. The Lloyd stereocampimeter showed central visual sparing from 0.25 to 1 degree, plotted with a 0.5 degree test object (table 1).

CASE 4.—M. S., a housewife aged 22, referred by Dr. W. H. LeFevre of Lancaster, Pa., was admitted to the neurosurgical service of the Hospital of the University of Pennsylvania on May 6, 1944, complaining of headache and vomiting. She had first noted a full, intermittent headache in the right occipital area nine months prior to admission. The headaches gradually increased in severity, and three months prior to her admission vomiting with the headache began. One month prior to admission she first noted blurring of vision. The following significant physical findings were elicited: A positive reaction to the Romberg test, with a tendency to stagger to the right; slight weakness of the right side of the face and bilateral papilledema of 2 D. The reflexes were generally hypotonic. The results of blood studies, urinalysis and serologic tests were normal. Roentgenograms of the skull revealed evidence of long-standing pressure. Preoperative studies of the visual fields showed partial right homonymous hemianopsia. On May 11, a ventriculographic study and left parieto-occipital osteoplastic craniotomy were carried out by Dr. Francis C. Grant. A large subcortical tumor was observed in the occipital area, and the left occipital lobe was amputated. The line of excision passed anterior to the parieto-occipital fissure and removed 0.75 cm. of the posterior horn of the left lateral ventricle. Pathologic diagnosis of the tumor revealed a metastatic rhabdomyosarcoma from the leg. The lobe measured 10 by 8 by 5 cm. and weighed 115 Gm.

Studies of the central visual field were made on July 16, 1946. Tests on the Bjerrum tangent screen with test objects of 1 and 3 mm. at 1 meter revealed preservation of central vision from 0.5 to 0.75 degree. Examination with the stereocampimeter of the fields plotted with a 0.25 degree white test object, showed central sparing up to 0.5 degree bilaterally (table 1).

CASE 5.—M. M., a housewife aged 34, referred by Dr. George Laws of Philadelphia, was admitted to the neurosurgical service of the Hospital of the University of Pennsylvania on Dec. 19, 1944. The patient complained of generalized headaches commencing two years prior to admission. After the onset of headache, the patient became aware of slight loss of memory and progressive loss of vision. One year prior to admission her vision became so poor that she was unable to read. The pateint was sent to the Presbyterian Hospital of Philadelphia, where studies aroused suspicion as to a possible tumor of the brain. The following pertinent physical signs were obtained: bilateral papilledema of 3 D., moderate anomia and alexia, slight mental dulness and loss of memory and generally hyperactive reflexes. Laboratory studies gave essentially normal results. Roentgenograms of the skull revealed evidence of increased intracranial pressure. Perimetric studies showed partial right homonymous hemianopsia. On December 22, a ventriculogram and left parieto-occipital osteoplastic craniotomy were carried out by Dr. Francis C. Grant, with exposure of a large meningioma, which appeared to arise from the tentorium. In order to remove the tumor, it was necessary to amputate the left occipital lobe. This was done at the parieto-occipital fissure, but the posterior horn of the ventricle was not opened. The tumor was completely extirpated, and most of the left half of the tentorium was excised. The patient made an uneventful recovery and was discharged on Jan. 26, 1945.

Examinations of the central visual field for the present check-up were done on July 17, 1946. The Bjerrum tangent screen showed preservation of central vision varying from 0.5 to 1.5 degrees, plotted with 1.5 and 3 mm. white test objects at 1 meter. The stereocampimeter revealed preservation of central vision from 0.5 to 1 degree, plotted with a 0.5 degree white test object (table 1).

CASE 6.—I. C., a white woman aged 52, referred by Dr. F. M. Dougherty, of Summit Hill, Pa., was admitted to the neurosurgical service of the Hospital of the University of Pennsylvania on Sept. 11, 1945, complaining of headache and vomiting. The headaches began in September 1944 and were bifrontal. Gradually they increased in intensity and were associated with vomiting and

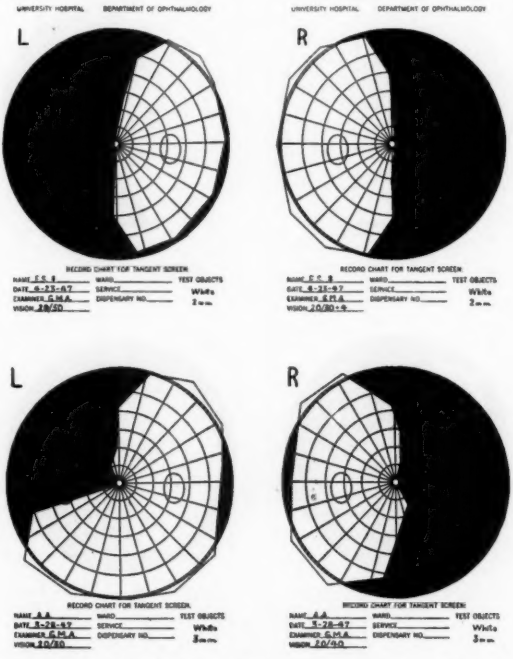

Fig. 4.—Central visual fields in 2 control cases with chiasmal lesions.

blurred vision. In January 1945 the headaches were associated with coma, occasionally lasting two or three days. The patient gradually became conscious of generalized weakness and loss of memory. The following significant physical signs were elicited: slight right hemiparesis and slight increase of the tendon reflexes on the right, and moderate adiadokokinesis of the left hand. The results of laboratory studies were essentially normal. Roentgenograms of the skull revealed a calcified lesion in the left parieto-occipital area. Preoperative examination of the peripheral fields revealed a right homonymous hemianopsia with sparing of central vision of approximately 6 degrees. On September 12, a left parieto-occipital osteoplastic craniotomy was performed, with the use of local anesthesia, by Dr. Henry Shenkin. A large tumor, involving the tentorium and the left occipital lobe, was revealed. The tumor was removed with part of the

tentorium. It was necessary to amputate the left occipital lobe. From the pathologic specimen the line of excision extended slightly anterior to the parieto-occipital fissure but did not involve the posterior horn of the ventricle. The occipital lobe and tumor weighed 240 Gm. and measured 10 by 6.5 by 7.5 cm. The pathologic diagnosis was that of meningioma.

Recent studies of the visual fields were carried out on July 17, 1946. The patient complained of difficulty in maintaining fixation, and for this reason only two central fields were obtained. The tangent screen showed sparing of central vision of from 0.75 to 1.25 degree. The stereocampimeter showed central sparing up to 1.5 degrees bilaterally.

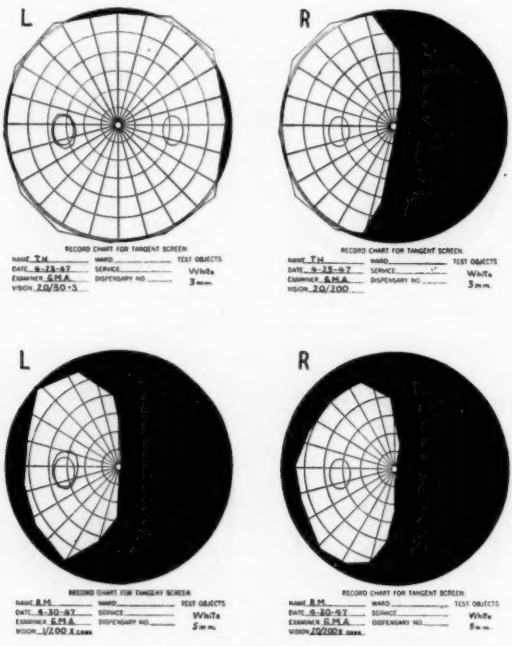

Fig. 5.—Central visual fields in 2 control cases with chiasmal lesions.

CONTROL CASES

It was impossible to obtain any cases of pure lesions of the optic tract for perimetric study at this time. However, we felt that chiasmal lesions would approximate a pure lesion of the tract and that such cases would serve as adequate controls. Accordingly, 8 cases of known mass lesions involving the optic chiasm were selected at random and the patients returned to the hospital for studies of the central fields. These were all made with the Bjerrum tangent screen, using different-sized test objects in repeated tests. The same close observation was employed, and the studies were carried out with the same screens and lighting conditions as those used in the cases of occipital lobectomy. The

TABLE 2.—*Preoperative and Postoperative Studies in 8 Control Cases with Chiasmal Lesions*

Case No.	Initials	Age	Color	Sex	Lesion	Vision at Time of Test	Date of Operation	Amount Preserved Central Vision in Postoperative Tangent Screen Test			Test Object	Date
1	.....	36	W	F	Pituitary adenoma (chromophobic)	O.D.:20/30+4, no cor. O.S.:20/50, no cor.	12/11/44	1:O.D. 1.25° O.S. 0.75°	2:O.D. 2.0° O.S. 0.5°	.....	2 and 5 mm. white at 1 meter	4/23/47
2	.....	66	W	F	Pituitary adenoma (chromophobic)	O.D.:20/40, no cor. O.S.:20/30, no cor.	12/ 8/38	1:O.D. 0.75° O.S. 1.5°	2:O.D. 1.0° O.S. 1.5°	3:O.D. 1.5° O.S. 1.5°	2, 3 and 5 mm. white at 1 meter	3/28/47
3	R. M.	50	W	F	Pituitary adenoma (chromophobic)	O.D.:20/200, no cor. O.S.: 1/200, no cor.	10/11/45	1:O.D. 1.0° O.S. 0.5°	2:O.D. 0.75° O.S. 1.0°	.....	2 and 5 mm. white at 1 meter	4/30/47
4	D. M.	24	W	F	Pituitary adenoma (chromophobic)	O.D.:20/200, no cor. O.S.:20/200, no cor.	3/13/46	1:O.D. 0.75° O.S. 0.75°	2:O.D. 1.0° O.S. 1.5°	3:O.D. 1.0° O.S. 0.75°	2 and 5 mm. white at 1 meter	4/11/47
5	T. H.	38	W	F	Craniopharyngioma	O.D.:20/200, no cor. O.S.:20/30+3, no cor.	1/ 9/47	1:O.D. 1.25° O.S. Normal	2:O.D. 1.0° O.S. Normal	.....	3 and 5 mm. white	4/23/47
6	C. L.	24	W	M	Craniopharyngioma	O.D.: Counts fingers, no cor. O.S.:20/15, no cor.	9/25/46	1:O.D. No test O.S. 0.75°	2:O.D. None O.S. 1.0°	....	2 and 5 mm. white at 1 meter	4/16/47
7	E. S.	17	W	M	Craniopharyngioma	O.D.: 0/ 0, no cor. O.S.:20/40, no cor.	11/10/41	1:O.D. No test O.S. 0.75°	2:O.D. None O.S. 0.75°	.....	2 and 5 mm. white at 1 meter	4/23/47
8	G. P.	39	N	M	Pituitary adenoma (chromophobic)	O.D.:20/30, no cor. O.S.:20/200, no cor.	3/13/45	1:O.D. 0.5° O.S. No test	2:O.D. 0.5° O.S. None	.....	2 and 3 mm. white	5/14/47

results of these studies are listed in table 2 and in figures 4 to 7. In no case was there any macular splitting, and the amount of central sparing was similar to that obtained in the occipital lobectomies.

COMMENT

The central fields revealed a limited degree of sparing of central vision in each case. In each case the findings of the Bjerrum tangent screen were closely corroborated by those of the Lloyd stereocampimeter. The lines of excision of the occipital lobes originated approximately at or anterior to the parieto-occipital fissure. The plane of section obviously varied in each case and in 2 cases did not pass through

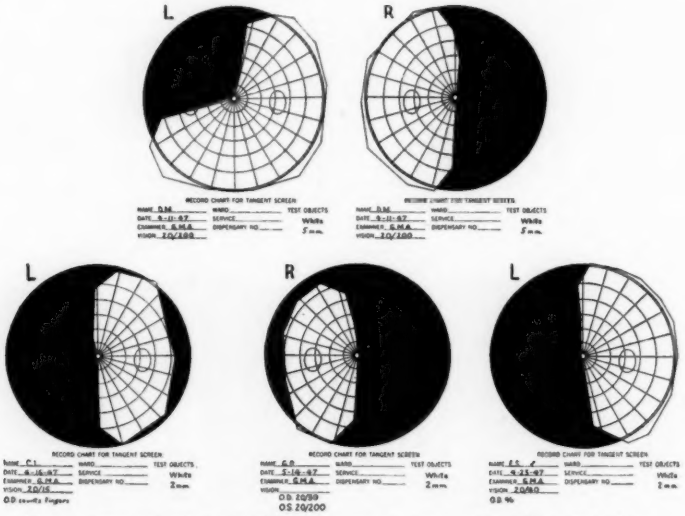

Fig. 6.—Central visual fields in 4 control cases with chiasmal lesions.

the posterior horn of the ventricle. In 5 cases the visual fields showed a slight meridional overlap of either the superior or the inferior quadrant on the blind side (table 1). Close examination seemed to reveal no obvious connection between the amount of preserved central vision and the plane of excision. Certain fundamental variations occur in every subject, and these cannot be overlooked in a test of this kind. Those of fundamental importance in this type of study are the distance the posterior horn extends into the occipital lobe, the anterior extent of the area striata [9] and the ability of the patient to maintain fixation.[10] We believe

9. Putnam, T. J., in discussion on Fox and German,[8a] pp. 821-826.

10. (*a*) Adler, F. H., and Fliegelman, M.: Influence of Fixation on the Visual Acuity, Arch. Ophth. **12**:475-483 (Oct.) 1943. (*b*) Traquair, H. M.: An Introduction to Clinical Perimetry, ed. 3, St. Louis, C. V. Mosby Company, 1940.

that the field findings were accurately repeated and checked in each case. Test objects of various sizes were used with each patient, and a close check on fixation was maintained. Lighting conditions and personnel were closely controlled, and all studies of fields were made by a member of the neurosurgical or the ophthalmologic staff.

In an excellent review of the subject of macular sparing, Putnam and Liebman [1b] stated that the fovea centralis covers an arc of approximately 2 degrees and the macula an arc of approximately 9 degrees. None of our findings can come under the heading of macular or foveal sparing according to these standards, since all the fields had less than 1.5 degrees of preserved central vision. There was an obvious individual variation in the amount of central vision retained in each case. This variation did not appear excessive as compared with the scale of measurement on the tangent screen or the stereocampimeter. Thus, in case 1 the variation was from 0.5 to 0.75 degree. In cases 5 and 6 the patients complained of difficulty in maintaining fixation and had a larger area of sparing with greater variation. This series of 6 cases was not large enough to serve as a basis for any definite conclusions, but it seemed that patients who were most recently operated on had the greatest difficulty in maintaining an accurate fixation, while maintenance of fixation was easier in cases in which the postoperative intervals were longer.

To explain the remaining areas of central vision by the theory of bilateral macular representation does not seem plausible, especially when there are small amounts of retained vision in the superior or inferior quadrants. Likewise, if there were a bilateral macular or foveal representation, the amount of spared vision should at least be equivalent in size to the arc of the fovea. The work of Hyndman,[8b] German and Brody [11] and Fox and German [8a] appears to have adequately refuted the assumption of a crossing through the splenium. However, as Walker [12] stated, the possibility of another, as yet unknown, crossing tract always remains. Working with chimpanzees and monkeys, von Bonin, Garol and McCulloch [13] were able to show that strychninization of area 17 leads only to firing within that area and to firing of the parastriate area. However, strychninization of the parastriate area or area 18 leads to the appearance of spikes within the parastriate area of the opposite hemisphere.

11. German, W. J., and Brody, B. S.: External Geniculate Bodies: Degenerative Studies Following Occipital Lobectomy, Arch. Neurol. & Psychiat. **43**:997-1006 (May) 1940.

12. Walker, A. E., in discussion on Halstead, Walker, and Bucy,[14] pp. 963-965.

13. von Bonin, G.; Garol, H. W., and McCulloch, W. S.: The Functional Organization of the Occipital Lobe, Biol. Symposia **7**:165-190, 1942.

Because we do not have histologic serial sections in each case to determine whether or not there was complete removal of the area striata, we cannot wholly exclude the possibility that a small remnant of striate cortex, sufficient to account for the amount of central vision preserved, remained anteriorly. Six cases, in the autopsy, in which there was no gross organic cerebral disease were picked at random. It was found that the posterior horn of the lateral ventricle had a rather definite anatomic relation to a coronal plane passing along the parieto-occipital fissure and through the junction with the calcarine fissure. There was a minimum distance of 1 cm. between this plane and the tip of the posterior horn. Even though the anterior projection of the area striata extends slightly beyond the intersection of the calcarine and the parieto-occipital fissure, we feel that excision through the posterior horn usually eliminates this remnant of visual cortex. It is not always necessary for the line of excision to pass through the posterior horn, however. For example, in the first case presented by Halstead, Walker and Bucy [14] no mention was made of exposing the posterior horn of the ventricle. Nevertheless, histologic serial sections revealed complete removal of the area striata. In Hyndman's case [8b] the resection was made "exactly tangent to the posterior tip of the corpus callosum," and the plane was passed through the common junction of the ventricular horns, exposing the glomus of the choroid plexus. However, in spite of section of the splenium, the patient still retained 2 degrees of central vision. Cases 5 and 6 reported by Fox and German [8a] are similar in the type of excision and in the results obtained. In their case 5, the occipital lobe was resected along a plane beginning 9 cm. anterior to the pole and extending through the junction of the posterior horn and the body of the ventricle. In their case 6 the plane of section passed just anterior to the junction of the calcarine and the occipitoparietal fissure. Apparently, in case 6 the posterior horn of the ventricle was not encountered. However, postoperative fields revealed sparing of 2 degrees of central vision in each case. Since histologic serial sections were not mentioned, it is again impossible to say definitely that all the area striata was removed. It is interesting to note that in these cases of Hyndman [8b] and of Fox and German,[8a] in which the line of excision varied, the sparing was small and essentially the same in all cases. Penfield and associates [8c] stated:

> Further, extensive removal of the occipital lobe including what seems to be all of the area striata can be carried out with sparing of macular vision.

We have chosen to interpret our own cases along somewhat similar lines, namely, that in each of the 6 cases there was complete removal or

14. Halstead, W. C.; Walker, A. E., and Bucy, P. C.: Sparing and Nonsparing of Macular Vision Associated with Occipital Lobectomy in Man, Arch. Ophth. **24**:948-966 (Nov.) 1940.

destruction of all visual cortex. In 4 of these cases the excision extended through the posterior horn of the ventricle. In all cases there was undoubtedly extension of necrosis beyond the actual line of excision. This was due to the use of a cutting Bovie current, with some interruption of the blood supply. This may have accounted for any remnant of visual cortex not actually included in the excised tissue.

In studies of the central fields after complete occipital lobectomy, Verhoeff [15] stated the belief that there is an apparent "macular" sparing due to loss of integration between the seeing fields and the blind fields. As a result of this loss of integration an eccentric retinal point is employed for fixation. Thus, the true field splits the macula, but in the central field tests the macular field appears to be more or less spared.

That a slightly eccentric retinal point serves almost as well for fixation as a normal one is apparent from the work of Adler and Meyer.[16] They showed that the fovea is not a point or a central cone but measures approximately 0.25 mm. in diameter. Furthermore, they showed that within this area sensitivity is the same and acuity depends on the number of retinal elements stimulated.

Bender and Kanzer [17] elaborated on the theory of a new functional macula, first presented by Fuchs [18] in 1920. After occipital lobectomy they found a small amount of preserved vision. This was not circular but consisted of an irregular, wavy vertical meridian. They stated the belief that occipital lobectomy led to the formation of a new functional macula with resulting "shifted" macula-splitting hemianopsia. According to Bender and Kanzer,[17]

> Fixation is brought about by directing the eye so that the object is in line with the visual axis, its image falling upon the fovea. During fixation the eye is not absolutely steady, but makes constant slight excursion, so that the fovea traverses the different parts of the object regarded and produces a synthetic picture. If there is a disturbance in central vision, such as occurs with a macula-splitting hemianopia, the retina of each eye will move into such a position as to obtain the best fixation possible, thus producing a new fovea.

Although in 4 of the 6 cases here presented, a slight overlap of the superior or inferior quadrant in the blind field was shown, there was no overlap in the others. This extension of the seeing field into the blind field was also present in several of the cases presented by Fox and German [8a] and was commented on at the time of presentation

15. Verhoeff, F. H.: A New Answer to the Question of Macular Sparing, Arch. Ophth. **30**:421-425 (Oct.) 1943.

16. Adler, F. H., and Meyer, G. P.: The Mechanism of the Fovea, Tr. Am. Ophth. Soc. **33**:266-280, 1935.

17. Bender, M. B., and Kanzer, M. G.: Dynamics of Homonymous Hemianopias and Preservation of Central Vision, Brain **62**:404-421, 1939.

18. Fuchs, W.: Untersuchungen über das Sehen der Hemianopika und Hemiamblyopiker, Ztscher. f. Psychol. u. Physiol. d. Sinnesorg. **84**:67-169, 1920.

by Mayer. However, in the 2 cases presented by Halstead, Walker and Bucy [14] and in those of Penfield, Evans and MacMillan [8c] this overlap was not evident. It is quite possible that this variation could occur in some cases after complete occipital lobectomy, but would not necessarily account for the central sparing in all cases, especially when a less marked eccentricity of the fovea would still be sufficient to alter fixation slightly and produce a slight central sparing. Verhoeff [15] made this point in stating that the more accurate the field tests are, the less retained central vision will be present.

Holmes [19] emphasized the plasticity of the visual cortex. He stressed that this development of the pseudofovea must be due to modification of function in the visual cortex and not to changes in the retina, which is a highly organized tissue. This is brought out by the fact that the sensitivity of the new point on the seeing side of the retina, employed for fixation, may eventually approximate that of the fovea.

The apparent sparing of central vision which remains in these cases can be explained by a minimal foveal shift and a slight variation in fixation which naturally accompanies it. As cited by Adler and Fliegelman [10a] and Traquair,[10b] the problem of complete fixation is a relative one. It is logical that the slight physiologic variation in fixation would be increased if any foveal eccentricity were present. From our observations it would appear that this ability to maintain fixation is increased in direct proportion to the postoperative time interval.

Kestenbaum [2] found homonymous hemianopsias with macular sparing of less than 3 degrees with lesions of both the tract and the optic radiation. He then concluded that sparing of less than 3 degrees has no topical significance and may be explained by a shift of the fixation toward the center of the preserved half of the macula. However, he also found cases of complete splitting in cases both of lesions of the optic tract and of the optic radiation.

Finally, we must consider the problem of interpretation of the visual fields in cases of small amounts of preserved central vision. Many authors have restricted by definition the term macular "sparing" to a preserved central fiield of not less than 10 degrees; others have called sparing similar to that presented here macular "splitting." With central vision varying from 0.25 to 1.5 degrees, the change might readily be interpreted as splitting of the central field if the area had not appeared consistently in repeated trials and had been found of similar magnitude in the control cases.

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19. Holmes, G.: The Organization of the Visual Cortex in Man, Proc. Roy. Soc. **132**:348-361, 1945.

The problem of splitting or sparing of the macula depends exclusively on the degree to which macular fibers are involved. In the optic tract the macular fibers are more exposed to pressure or to a lesion from the outside than in the optic radiation, where they begin to fan out. Hence, the tendency to splitting will be greater with lesions of the optic tract than with those of the radiation.

The clinical experience that lesions of the optic radiation are characterized by a tendency to macular "sparing" and those of the optic tract to "splitting" still holds true if the standard methods of perimetry only are used.

From the physiologic point of view, the heated debate about splitting and sparing of the field of central vision has lost its meaning. Neither does true splitting seem to be present in the average case of lesion of the tract unless the term macular "splitting" is used loosely to include those cases with preserved central vision of less than 2 degrees.

Thus ends also the search for a valid theory to explain macular sparing in cases of lesions of the occipital lobe. The problem has shrunken to the question of the extent of the lesion. Obviously, the lesion may be small and may not extend far enough forward or backward to affect all the macular fibers. Or it may be large enough to involve the entire striate area, and yet some central vision may be present owing to formation of a new functional fovea.

SUMMARY

Six cases of occipital lobectomy are presented in detail, and their results with regard to preservation of central vision are summarized.

The various theories advanced to explain macular sparing are discussed. Previous series of occipital lobectomy are reviewed with reference to the present status of the doctrine of cortical localization of the macula.

It is concluded that there is no evidence to support a theory of bilateral macular or foveal representation.

Preservation of central vision ranging from 0.25 to 1.5 degrees was observed in our patients with occipital lobectomy. However, the same degree of macular sparing was found in 8 control patients with chiasmal lesions. It was felt that the situation and extent of the lesion in the optic pathways, as well as a minimal eccentricity of the fovea with slight physiologic variation in fixation, explain the results most satisfactorily.

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ALLESTHESIA AND DISTURBANCE OF THE BODY SCHEME

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CHANGES in behavior produced by focal cerebral lesions in man usually arouse the interest of neurologists and psychologists. By contrast, alterations in behavior which appear in the course of a diffuse degenerative process in the brain are only too often dismissed as instances of "simple deterioration" or "dementia." This neglect persists despite Goldstein's [1] repeated warnings that studies of cases of so-called organic deterioration may add to the understanding of intellectual function in normal and abnormal states.

It is true that the clinical picture of many of the psychoses associated with degenerative disease of the brain may appear complex, or even bizzare, and that the patients are difficult to test. Yet simplicity and complexity are relative matters depending on the investigator's point of view. If one looks for clearcut losses of specific functions, these psychoses may seem unrevealing, because the patients manifest alterations in many different performance fields. For those, however, who consider the varied symptoms after cerebral lesion as interrelated,[1a] a mental syndrome found in patients with disease of the brain is far from bizzare. From such a point of view, the changes in performance are not specific but reflect a general alteration in behavior which can be recognized in every individual symptom.

With the difference in theoretic viewpoints there frequently goes a difference in the methods of examing a patient. The proponent of

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Work aided in part by a grant from the fellowship fund of the Emanuel Libman Foundation.

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Presented (with demonstration of the patient) at the combined meeting of the Section of Neurology and Psychiatry of the New York Academy of Medicine and the New York Neurological Society, Jan. 13, 1948.

1. (*a*) Goldstein, K.: The Organism, New York, American Book Company, 1939, pp. 17 and 533. (*b*) Goldstein, K., and Katz, S. E.: The Psychopathology of Pick's Disease, Arch. Neurol. & Psychiat. **38**:473-490, (Sept.) 1937.

specificity may limit himself to a few tests which tap areas of maximal dysfunction. The proponent of generality, however, will try to sample the patient's performance in a great number of fields. He realizes that what appears to be a specific loss of a single function may be due to the use of one single test. Moreover, not only does he search for evidence of total loss of specific abilities (negative symptoms); he is equally intent to uncover the possible alterations in mode of functioning (positive symptoms) which his patient may show.[2] Such alterations may manifest themselves, not in failure in certain tasks, but in the adoption of round-about procedures for their solution.[3]

If one studies the behavior of patients with "organic deterioration" in this way, these patients can be shown to present a considerable degree of generality of dysfunction.[4] To be sure, evidence from such cases, in which the pathologic changes in the structures are admittedly diffuse, cannot be used to settle the issue whether generality or specificity of symptoms is the rule in all cases of cerebral lesion. However, these cases illustrate the patterns of dysfunction that may appear after massive cerebral involvement. Moreover, cases like the one about to be described may show unusual yet consistent forms of sensory and motor disturbances which are seldom observed unless there is extensive damage to the brain.

REPORT OF A CASE

H. S., a 52 year old right-handed carpenter, was admitted to Bellevue Psychiatric Hospital on Aug. 8, 1947 because of progressive "mental deterioration." In 1944, ten days following a ureteral transplant performed for carcinoma of the bladder, there suddenly developed a paresis of the left upper extremity, confusion and mumbling speech. He improved, and within a few weeks his wife could not notice any disability. Two years later, in 1946, the symptoms reappeared for a short period. A third episode of weakness in the right hand occurred just prior to admission. His wife reported that during the past two years his behavior had become increasingly peculiar. She stated, "He doesn't know the back from the front of his own person or the right from the wrong side of things. He puts his pants on upside down."

On admission, the patient appeared well nourished and well developed. On routine psychiatric examination, he seemed to present an "organic mental syndrome" characterized by "disorientation in time and place," "defects in recent and remote memory" and "emotional instability." He was cooperative and his

2. Jackson, J. H.: Selected Writings (ed. by James Taylor) London, Hodder & Stoughton, Ltd., 1931, vol. 1, pp. 14 and 500; ibid. 1932, vol. 2, pp. 8 and 510. Goldstein.[1a]

3. Goldstein, K., and Scheerer, M.: Abstract and Concrete Behavior: An Experimental Study with Special Tests, Psychol. Monogr. **53**: (2) 1-151, 1941.

4. Bender, M. B.; Wortis, S. B., and Cramer, J.: Organic Mental Syndrome with Phenomena of Extinction and Allesthesia, Arch. Neurol. & Psychiat. **59**: 273-291 (March) 1948.

mood was usually affable. He seemed to have some insight into his condition so far as he was aware of somatic difficulties; but he tended to ascribe these difficulties to an uncooperative environment. This "shifting of blame" led some members of the staff to consider the patient as "mildly paranoid." However, there was nothing systematic about the patient's accusations.

Neurologically, he presented evidence of bilateral pyramidal tract involvement. This was more prominent on the left side. There were positive Hoffmann and inconstant Babinski and Chaddock signs. Abdominal and cremasteric reflexes were absent. There were motor weakness and increased tonus in the left upper extremity. The cranial nerves were grossly intact. Wassermann reactions of the blood and spinal fluid were negative. The spinal fluid was under normal pressure and had no increase in cells but had a total protein content of 160 mg. per hundred cubic centimeters. The electroencephalogram recorded diffuse abnormality. A pneumoencephalogram revealed considerable amounts of air over the convexities of both cerebral hemispheres and in the entire ventricular system. These findings were interpreted as consistent with a diagnosis of diffuse degenerative encephalopathy.

The patient was studied in great detail over a period of four months. A record of some of the observations follows.

I. Disturbances in Sensation.—A. Cutaneous Sensations: On routine testing there appeared to be hypalgesia on the left side. Light touch and temperature were equally well perceived on the two sides, but remarkable responses were obtained when the patient was asked to indicate the site of stimulation. A brief stimulus applied to the face, upper extremity, trunk or abdomen on the right side was localized by the patient to a homologous area on his left side. This false localization of sensation—allesthesia—was noted for all cutaneous modalities. Such allesthesia—that is, localization to the opposite side of the body—was the rule. Occasionally the mislocalization was less extensive; even then, it was consistently to the left of the point actually stimulated (fig. 1*A*). By contrast, stimulation on the left side of the body was always correctly localized (i. e., errors on the left did not exceed the errors made by normal controls).

Allesthesia was also present on bilateral stimulation. When tactile stimuli were simultaneously presented to both sides of the patient's body (right and left), the patient usually reported that he felt two contacts, but localized both to his left side. Occasionally, however, he reported only one contact and localized this contact to his left side. In these instances, the patient manifested the phenomenon of extinction.[5]

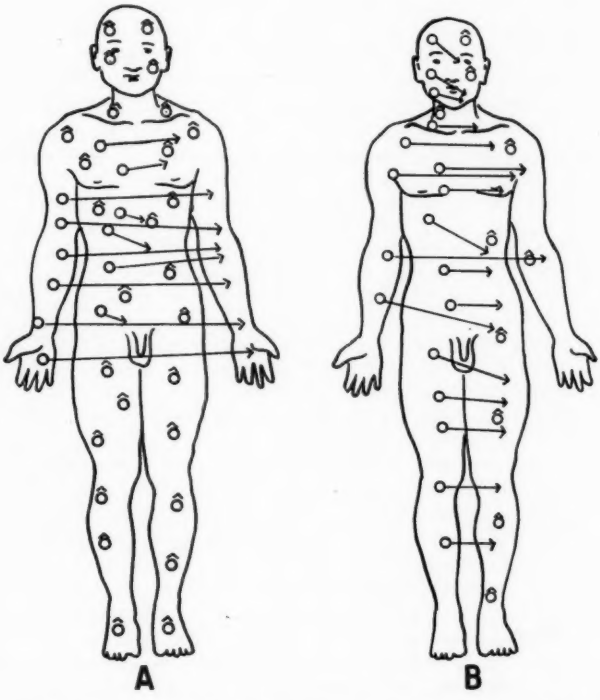
The cutaneous allesthesia appeared regardless of whether the patient attempted to localize the stimulus by verbal description or by pointing. When he pointed, it made no difference whether he had his eyes open or closed, or whether he was allowed or forbidden to touch the presumed site of stimulation. The extent of the cutaneous allesthesia, however, was influenced by at least three factors.

(*a*) One of these factors was the condition of the patient and the course of his illness. During the first week following admission, when the neurologic and psychiatric symptoms were most marked, cutaneous allesthesia or the erroneous

5. Bender, M. B., and Teuber, H. L.: Phenomena of Fluctuation, Extinction and Completion in Visual Perception, Arch. Neurol. & Psychiat. **55**:627-658 (June) 1946.

localization of stimuli from the right to the left could be demonstrated over the entire right half of the patient's body except for the right lower extremity. During the second week, allesthesia on stimulation of the face became inconstant, and in the third week it could no longer be elicited. Throughout the entire period, allesthesia could be demonstrated on the right side of the trunk and the right upper extremity.

(*b*) A second factor in the appearance of allesthesia was the mode and strength of stimulation. Pain was the only cutaneous modality in which duration and intensity of stimulation made a difference in the patient's response. Alles-



Pattern of erroneous localization of tactile stimuli (allesthesia) before and after injection of amobarbital. Circles indicate the site of application of the stimulus (pinprick); the head of the arrow, the site of localization by the patient. A caret above the circle indicates localization within normal limits. *A,* status prior to injection of amobarbital sodium. Allesthesia is noted on stimulation of the right upper extremity and to a lesser extent on stimulation of the right half of the trunk. *B,* status under the influence of amobarbital sodium. Allesthesia not only is elicited in the previously indicated regions but may also be noted on stimulation of the right half of the face and the right lower extremity.

thesia was consistently evident when a painful stimulation such as pinprick was brief or even repetitive. However, when the pinpoint was firmly and steadily applied and the patient manifested reactions indicative of pain, the localization

was correct. In all other cutaneous modalities there was erroneous localization, whether the stimulation was light or intense, brief or prolonged.

(*c*) A third factor which influenced the allesthesia was the effect of amobarbital sodium (amytal sodium®). This drug was administered at a time when the allesthesia was present only in the trunk and arm. It should be recalled that it had never been demonstrated for the right leg. After amobarbital sodium had been injected intravenously to the point of appearance of ocular nystagmus (approximately 0.3 Gm. in one hundred and twenty seconds), allesthesia became evident over the entire right half of the body, including the face—where it had been observed consistently only in the first week—and the right leg—where it had never been observed (fig. 1*B*). This increase in the areas in which allesthesia could be demonstrated lasted approximately twenty minutes. At the end of this period the pattern of localization returned to the preinjection state.

B. Visual Perception: On confrontation, perimetry and tangent screen examinations the patient's visual fields appeared to be intact for form and color. Occasionally, a concentric contraction of the fields was noted, but this contraction was inconstant. However, special tests revealed that the patient had difficulty in localizing objects in his visual space. When test objects were presented anywhere in his right field of vision, he insisted that they were "next to" or "on top of" the point on which he was fixating. The same tendency appeared when the patient tried to reach for objects exhibited in the right field of vision; no matter where the object actually was, he groped for it near the fixation point.

When test objects were simultaneously presented in each of the lateral fields (right and left) while the patient looked straight ahead, he often stated that he saw both objects in his left field. Such apparent displacement of a visual image from one side to the other is known as optic allesthesia. The frequency with which optic allesthesia could be elicited in this patient seemed to vary with the duration of the exposure. When the test objects were briefly presented, the allesthesia was more apparent than when the exposure was prolonged.

C. Auditory Perception: Allesthesia was also noted when hearing was tested. All sounds originating above the patient's head, behind him or to his right were localized as coming from his left side. When noises were produced simultaneously on each side of his head he stated that "all the noise" came from his left side. He made no significant errors in localization when the sounds originated on his left.

This auditory allesthesia was constant during the first week of observation, inconsistent during the second and could not be demonstrated after the third. However, under the influence of amobarbital sodium, as we have described, the auditory allesthesia once more appeared and lasted approximately twenty minutes.

II. Alterations in Motor Pattern.—In his motor behavior, the patient showed a tendency to prefer his left side (levotendency). This levotendency was manifested as follows: The patient stood with head and trunk tilted to the left. The right side of his body was held slightly advanced and on walking seemed to have a tendency to rotate forward and around to the left. When asked to reverse his direction in walking, he usually turned to the left. He did this even when obstacles were placed in his way to make turning to the left side difficult. Anyone watching his behavior got the impression that the patient was "pulled" to the left. Similar systematic motor deviations appeared when the patient was asked to point. Whenever he tried to localize a cutaneous stimulus applied to the right forearm, he went through a characteristic sequence. The left hand was slowly

brought to the midline and held there for a moment. He then dropped his left hand, raised his right and pointed to his left forearm.

The patient was able to write the first one or two letters of his name. The remaining letters, however, were written over the first letters so that the patient ended with an illegible compressed scrawl. He expressed frustration and volunteered, "I don't know what's wrong; it always goes to the left, and then there's no space left."

III. Disorientation in Space.—A. Orientation on the Patient's Own Body: The patient could locate parts of his body which were in the midline, such as head, nose, mouth, chest, abdomen, umbilicus and penis. However, he had difficulties with paired structures. On attempting to locate his eyes or his ears he consistently overshot his mark to the left. He first pointed to his left eye or left ear and then pointed into space to the left of his head to indicate the site of the other eye or ear. This tendency to localize the eyes and ears farther to his left was found only as long as there was allesthesia for various stimuli applied to the face.

The patient was able to locate his wrist, elbow and shoulder. However, he did so by pointing them out on his left side. When the examiner insisted that he had more than one wrist or elbow, the patient agreed but tried to locate the "other" wrist just below his left wrist and the "other" elbow somewhere near his left elbow or, occasionally, behind his left shoulder. He was unable to find any of these parts of the body on others. This difficulty was present even after the examiner's wrist or elbow had been pointed out to the patient. He could not identify individual fingers on himself or on others.

B. General Spatial Orientation: The patient was unable to distinguish "right" from "left" with reference to objects in space, just as he was unable to distinguish right and left with reference to parts of his own body. He had a semblance of orientation in terms of "good" side and "bad" side; when asked to point to his bad side he touched his left wrist, and yet he could not find the whereabouts of any other part of the body on his presumably "bad" side. In fact, he had no concept of laterality. He seemed unable to comprehend the terms upward, downward, in front of, in back of, to the side of and the like. He was presented with manikins and drawings in which parts of the body had been deliberately misplaced (e. g., eyes placed outside the head, legs drawn upside down), and he seemed to be unable to find anything wrong with any of these pictures. Even when a correct figure was placed beside a distorted one, he denied that there was any difference between the two.

This difficulty with spatial relations may have been a factor in the patient's particular form of apraxia. Often, he seemed incapable of manipulating objects in a manner adequate for their use, and this inability appeared most clearly when it was necessary for him to orient the particular objects in space. When a pencil was handed to him, he sometimes attempted to write with the blunt end, or he would try to place a milk bottle upside down on the table or to put the burning end of a cigaret into his mouth. He was unable to put on any article of clothing. In these situations he showed little insight. He did not even revert to trial and error behavior but expressed disgust with his failure and insisted that the pencil was bad, the bottle poorly constructed or the pajamas "no good."

During the period of observation, he was given series of tests involving the copying of outline figures by drawing, or with matchsticks, such as the visual

retention test of Benton [6] and the stick test of Goldstein and Scheerer.[3] He was quite unable to copy simple geometric figures, such as a triangle or square, but he insisted that the jumble of lines or sticks he had put together was identical with the examiner's model.

IV. Intellectual Changes.—A. Routine Psychologic Tests [7]: In spite of the described changes in performance, the psychologists felt that the patient's personality structure was essentially intact. On the verbal subtests of the Wechsler-Bellevue Scale,[8] he earned an intelligence quotient of 79, representing a borderline normal performance. On the other hand, he showed total inability to deal effectively with the "visuoconstructive" subtests of the scale, such as object assembly, block design, picture arrangement and picture completion. Routine Rorschach examination was reported as showing "organic disease of the brain," signalized, for instance, in poor "visuospatial organization." Here again it was stressed that the patient's personality seemed remarkably well preserved.

B. Special Psychologic Tests: On tests performed in the neurologic laboratory, such as the grouping and sorting tests of Goldstein and his students,[3] the patient showed rigid perseveration in any given set. His behavior was of the sort that led Goldstein to speak in such cases of extreme "concreteness" of thought processes. He seemed to have almost insuperable difficulties in considering more than one aspect of any situation at a time. In particular, he seemed unable to conceive of purely imaginary or fictitious situations. He could not knock at an imaginary door and tended to interpret proverbs and figures of speech in an inappropriately literal fashion.[9] We praised him during one of the many examinations: "This time you hit the nail right on the head," and he retorted: "A nail I always hit on the head—I used to be a carpenter." This peculiar obtuseness went so far that he was suspected at times of having some dysphasia or agnosia. Actually, his language difficulties did not present anything beyond the general "perseveration," rigidity, and one-track-mindedness which he showed in his intellectual performance.

The semblance of agnosia, at least during the first admission, could probably also be understood in terms of his general intellectual impairment. He recognized snapshots and outline drawings of objects and persons, but he held the pictures in any position—right side up or upside down—without noticing any difference in orientation. Occasionally, he commented on the pictorial material in such a way that we doubted whether he could distinguish between the pictures and the objects they represented. On some of the cards in the Thematic Apperception Test,[10] he pushed his nails against the margin of the photograph, as though to lift it from its white cardboard background. In a special test for visual agnosia, he looked at the photograph of a wooden fence and declared it was part of a house. We asked, "Can you look into the house? Can you tell us what's inside?" and the patient turned the card over, slowly and deliberately, and searched in back of the picture.

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6. Benton, A. L.: A Visual Retention Test for Clinical Use, Arch. Neurol. & Psychiat. **54**:212-216 (Sept.) 1945.

7. These tests were given by the ward psychologists under the dircetion of Dr. David Wechsler.

8. Wechsler, D.: The Measurement of Adult Intelligence, ed. 3, Baltimore, Williams & Wilkins, 1944.

9. Goldstein, K.: Ueber Aphasie, Schweiz, Arch f. Neurol. u. Psychiat. **19**:1-68, 1926.

10. Murray, H. A.: Thematic Apperception Test Manual, Cambridge, Harvard University Press, 1943.

He had difficulties in reading, but these difficulties reduced themselves to an inability to "keep his place" on the printed page. He skipped single letters, words and entire lines, and his gaze invariably directed itself to the lower left corner of the page. That this was not "simple alexia" could be shown by covering all words in the context but one.[11] Under such conditions, the patient could read and comprehend. Again, the difficulty in dealing with relations in space seemed to be an important common factor in his failures in different performance fields.

COMMENT

In conventional clinical terms the case recorded here is one of "mental deterioration" or of "disorientation" which impaired the patient's ability to deal with his own body as well as with objects in his surroundings. Yet such a summary in terms of "negative" symptoms seems insufficient to describe the patient's performance. The patient also had "positive" symptoms. The most prominent of these was allesthesia, or erroneous referral of sensations from one side of the body to the other, on cutaneous, visual and auditory stimulations. The allesthesia was manifested in a consistent tendency to attribute stimulation which actually impinged on his right side to homologous areas on his left. There were parallel tendencies toward displacements from right to left in the motor sphere (levotendency). On sitting and standing, he tilted his head and trunk to the left; on walking, he rotated the right shoulder forward and to the left. He always turned to his left side. Subjective displacement of sensory impressions, as well as the objective distortion of the patient's motor pattern, were in the same direction in space. Both the motor and the sensory disturbances were exacerbated by the injection of amobarbital sodium. There was an increased tilt to the left, and sensations over the entire right side of the body were referred to the left.[12]

11. Rothmann, E.: Untersuchung eines Falles von umschriebener Hirnschädigung mit Störungen auf verschiedensten Leistungsgebieten, Schweiz. Arch. f. Neurol u. Psychiat. **33**:3-51, 1933.

12. Since the levotendency in the patient's motor behavior was marked, one should consider the possibility that the allesthesia was merely a secondary effect. One might say that the patient correctly perceived stimuli on his right (i. e., perceived them as really being on that side) but could not prevent himself from pointing to the wrong (left) side of his body because of the persistent levotendency in his motorium. However, he made the same errors when he did not point but indicated the presumed site of stimulation verbally. This suggests that in this case the perceptual component (allesthesia) was the major factor in producing the patient's mislocalizations. Yet there are undoubtedly many cases of allesthesia in which it is impossible to make any distinction between an apparent displacement of sensations (perceptual component) and an erroneous pointing (due to dextrotendency or levotendency). Such cases demonstrate the precarious nature of the dichotomy between apraxia and agnosia, since we cannot meaningfully separate the sensory and motor component of the patient's performance.

Instances of allesthesia after cerebral damage have been described by Kramer,[13] Pick,[14] Herrman and Pötzl[15] and, in particularly detailed reports, by Schilder,[16] who emphasized the possible theoretic significance of these cases for our understanding of the cerebral basis of somesthetic functions. In all previously reported cases, there was evidence of massive bilateral lesions, just as in the present case. An unusual feature of the present case, however, was the patient's persistent tendency to refer sensations to the formerly hemiplegic side. By contrast, systematic referral from the defective to the "better" side of the body has been characteristic of the cases which have been reported earlier.[17]

The patient's particular insistence on his "bad" side in the present case makes it difficult to interpret the disturbance in a simple teleologic fashion. In the usual forms of anosognosia (the denial of paralysis) and autotopagnosia (the denial of the presence of the impaired part of the body) the patient may be said to exclude the impaired portions of his organism from his own awareness. The opposite seemed to have happened in the present case; everything "pulled" the patient, as he put it, to his left (or comparatively more impaired) side.

It should be noted that the disorder which on the surface appeared to be an instance of simple confusion was, in fact, "orderly" or patterned. Distortions in the patient's conception of his own body conformed to the abnormal motor tendencies, and there was a corresponding disorientation of outer space.[18]

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13. Kramer, F.: Alloästhesie und fehlende Wahrnehmung der gelähmten Körperhälfte, Ztschr. f. d. ges. Neurol. u. Psychiat. **11**:379, 1915.

14. Pick, A.: Studien zur Hirnpathologie und Psychologie, Berlin, S. Karger, 1908; Störung der Orientierung am eigenen Körper, Psychol. Forsch. **1**:303-318, 1922.

15. Herrman, G., and Pötzl, O.: Die optische Alloästhesie: Studien zur Psychopathologie der Raumbildung, Berlin, S. Karger, 1928.

16. Schilder, P.: The Image and Appearance of the Human Body (Psyche Monographs, No. 4), London, Kegan Paul, Trench, Trubner & Co., Ltd., 1935.

17. Schilder[16] (p. 305 and following) described a patient who claimed to feel a " second touch" on the right (paretic) side of her body after a tactile stimulus had been applied four to ten seconds earlier to her left side.

18. If one assumes that the disturbance in the body scheme was the principal factor in the disorder, one might attempt to describe the interrelation of symptoms in the following way: The patient's own body provided the system of coordinates for the objects he had to deal with. Since this system was distorted, the environment assumed for him equally distorted aspects. Proper manipulation of things became impossible, since he had lost the common ability to move in a quasigeometric world with orderly spatial relations.[18a] To him, each stimulus object seemed to have its own center of gravity; it was no longer anchored in a network of abstract spatial relations. Thus, all stimuli applied directly to his body surface, except for protracted painful stimuli, yielded, one might say, to the abnormal tonic pull (levotendency) toward the patient's impaired side.

This case would thus seem to illustrate a close relationship between what has been called the body image [19] and the ability to orient oneself in space. Distortions in one tend to entail disturbances in the other, and the resultant total disorder is far from unpredictable, if the patient is submitted to a sufficient number of tests. The reward of such efforts may be the gradual elucidation of patterns of malfunctioning. Cases of this sort obviously do not contribute anything to a discrete localization of cerebral function, but the description of patterns of malfunctioning seems to us a necessary intermediate step which must be taken before one tries to uncover the relationships between cerebral structure and function.

SUMMARY

In a case of diffuse encephalopathy, the outstanding symptom was allesthesia, or consistent erroneous referral of sensations from one side of the body to the other. Cutaneous, auditory and visual stimuli impinging on the patient's right side were localized by him to approximately homologous areas on the left. There were associated motor and intellectual disturbances suggesting a syndrome in which changes in the body scheme and inability to deal with spatial relations formed the principal features. It is pointed out that this patient's behavior is not adequately described by the term "simple deterioration." Despite the severity of alterations in sensory, motor and intellectual functions, the disorder of such patients may be patterned and their performance predictable.

DISCUSSION

DR. KURT GOLDSTEIN, New York: This is a very interesting case, valuable from the practical and theoretic concepts of brain function. Regardless of whether one is dealing with multiple defects or whether all the disturbances may be secondary to a single impairment, one should start with the consideration of a single phenomenon, allesthesia, or the displacement of stimuli impinging on the right side to the left side of the body. I am not sure whether one has to consider this displacement as a disturbance of the cerebellofrontal system or as a real allesthesia, though I am inclined to explain some of the phenomena by a lesion of the cerebellofrontal system. The assumption of real allesthesia meets difficulty because the dislocation occurs only on stimulation of one side. Besides, the term allesthesia is simply descriptive and does not help one to understand the underlying functional defect. If the dislocation is not an expression of a dysfunction of the cerebellofrontal system, another explanation should be considered—namely, the assumption that the patient's symptoms are due to the phenomenon that stimuli impinging on a part of the body with increased threshold of sensitivity are dislocated to a part with lower threshold if, by so doing, a useful perception can be achieved. I should like to point here to the dislocation in hemianopsia toward the fovea. The same occurs in stimulation of skin points toward the place of greater sensitivity. In this respect it is worthy of note that

19. Head, H.: Studies in Neurology, London, Hodder & Stoughton, Ltd., 1920.

the patient dislocated only slight stimuli—i. e., such as apparently were not well perceived—but when the "pinprick was firmly and steadily" applied, perception occurred at the stimulated place. This explanation could be supported by the observation that the patient's dislocations of objects presented in the right field of vision were experienced "next to" or "on top of" the fixation point and that he groped for the object always nearer to the fixation point. It would also agree with the fact that the dislocation disappeared with improvement of the condition, and it clarifies the effect of amobarbital sodium, which heightens the effect on the thresholds, which on the left side did not hinder correct perception.

The second interesting symptom of the patient is his impairment of abstract attitude. Whether the dislocation and impairment of abstraction are related to each other is difficult to say. I consider it possible that the two groups of symptoms occurred together because they are related to a somewhat similar localization of the lesion—namely, the frontal lobes. Maybe, indeed, the patient has various lesions. I should guess that there is diffuse damage of the left hemisphere, including the left frontal lobe, which has increased the thresholds and produced the impairment of abstraction, while the dislocation to the left side, the pull to this side and the damage of the body image may be due to a lesion in the right frontal lobe.

Dr. Lauretta Bender, New York: This is a very rewarding patient to have studied, who interested me from a number of points of view. I have been interested in hearing Dr. Goldstein discuss the case, because I should like to discuss it from a different viewpoint. Dr. Schilder's early work on the body image (Schilder, P.: The Image and Appearance of the Human Body, Psyche Monographs, London, George Routledge & Sons, Ltd., and Kegan Paul, Trench, Trubner & Co., Ltd., 1935) was on the basis of this type of case—with left-right difficulties and allesthesia. He did not have a case quite like this, and it presents some special problems because of the additional factors brought in. He did raise the question as to whether the crossover from one side to the other was related to spinal tract crossover as well as the psychologic body image problem, and he always noted in the cases with which he was familiar that there was a transfer from the poor to the good side. It is clear in the present case, since the visual and the auditory problems are brought in and the motor impulses are there, as well as concrete thinking and difficulties in the relation in space and the projection on the body image of other persons, that this is more than a localization problem. It is a much more complicated problem. I think that it can all be explained on certain fundamental principles, and I hope that you will excuse me if I refer to my own experience with children. In children one sees instances, either in psychoses or in different kinds of encephalopathies, in which there is an impulse to turn around the longitudinal axis. Incidentally, Dr. Schilder did attempt to discuss the allesthesias with the possibility that they could be explained by the turning on the longitudinal axis. Children give way to their impulses freely, and consequently we have longitudinal turning or whirling in children as a regressive neurologic phenomenon. In such children we have an expansion of the body image problem. Adults are more likely to resist pathologic impulses; in the posture of this man one sees the tendency to turn on the longitudinal axis from the right to the left, but one must increase his motor impulses by walking or telling him to turn before he gives way to the impulse. I think that all the disturbances in the perceptual field can be analyzed on the basis of this impulse to turn and the resulting distortion in body image concept from the right to the left; the individual, however, restricts it, and there is a contraction of the body image. We are told that cutaneous sensation is not always projected to the

homologous point, and in this case it frequently fell short of that. If you think of many other points in the sensory examination, you will find that they can be explained on the basis of contraction of the body image and contraction of the environment. Interestingly, although this patient cannot orient himself in space in abstract terms, he does so in concrete terms. One gets the feeling that the ceiling and the floor are too near, that right and left are too near. Certainly it is clear that in pointing to the parts of Dr. Shapiro's body the patient said that the hand was up near the neck, and he pointed to it there. One gets in this the impression that in this man there is an impulse to turn on the longitudinal axis, but, owing to the restriction of his motor impulses, the rigidity of his intellectual functions and the lack of plasticity of the body image, there is a contraction instead of an expansion of the body image. Interestingly, in children we get the expansion, and we can see it in their fantasies and fears. It is always the hands which are most prominent. Children who are suffering from these anxieties relating to turning impulses all complain that their hands are getting too big and too far away from them and that the walls are contracting on them, whereas their body is expanding, and they are frightened. It is interesting in this adult that when all the other allesthetic signs disappear allesthesia still remains in the hands.

There is one other point that I want to bring out. The barbital which stimulates the vestibular areas leads to an increase of the impulses to turn, and this fact would explain the increase in symptoms in this man or the tendency to expand the otherwise contracting body image. The thinking disturbance is interesting in this regard, too; even the man's name is contracted.

I am inclined to explain the whole picture on the basis of the motor impulse to turn from the right to the left, an intellectual contraction in thinking, and a contraction of the body image and the spatial environment.

DR. ALLEN S. MARINER: I should like to ask Dr. Lauretta Bender a question. When you say the whole syndrome can be explained by contraction of both personal and extrapersonal space, do you leave this as an observed phenomenon? In other words, is that an explanation, or is it a gathering together of all the observations under a common head? If it is an explanation, can you carry it back a bit further?

DR. LAURETTA BENDER: How do I get my thinking? I cannot go so far back. To me my statements explain the syndrome in psychologic terms.

DR. ALLEN S. MARINER: Perhaps I should ask more directly: Why is there a contraction?

DR. LAURETTA BENDER: I tried to account for the contraction by assuming that in children there is a tendency to expand because children give way to their impulses and that as they grow older there are tendencies which lead to restrictions. There is evidence in this man of expansion and contraction; the impulses become piled up; in this patient there is rigidity due to the pathologic state, and rigidity tends toward contraction because of the pathologic process which comes out of the situation. There are concrete thinking and diminution of energy as the process continues.

DR. MORRIS B. BENDER: Thus far we have no explanations for the phenomena observed in the patient presented tonight. We are still in the process of studying this and other cases. On numerous examinations and with different methods of testing we found that this man showed a definite pattern in his reactions to sensory stimuli and in his motor behavior.

Dr. Goldstein implied that allesthesia results from the patient's tendency to displace a sensation to a point or an area in which there is better performance. This explanation is attractive, but there are a few observations which seem not to support Dr. Goldstein's contention. Our patient complained that his left side was "bad."

DR. KURT GOLDSTEIN: Are you sure he is to be believed?

DR. M. B. BENDER: This is what he reported. The patient consistently said that he felt "less" on the left side. Moreover, the history and examination revealed impaired motor and sensory functions in his left hand. His performance was poor in the left hand when compared with that of the right.

DR. KURT GOLDSTEIN: The hemianopsia is on the right, is it not?

DR. M. B. BENDER: At present the defects in his field of vision are bilateral but apparently greater on the right side. When this patient was first seen by us, he had a fairly concentric contraction of his visual fields—no hemianopsia or hemiamblyopia. This man is only 1 of a series of 5 patients with allesthesia whom we have studied at Bellevue Psychiatric and Mount Sinai hospitals. In another patient (J. G.) the allesthesia was most pronounced in vision. When an object was placed in the periphery of the right field of vision, he localized it either at the fixation point or slightly to the left of it. It seems that this patient displaced the target to an area of best performance—namely the fixation point. However, this was not always the case. When two objects were exposed, each in the periphery of one half field of vision, J. G. either saw the object on the left side alone (phenomenon of extinction) or saw both objects, but localized them both in the periphery of the left half-field of vision (allesthesia). In these circumstances the displacement was not to the fixation point (area of best performance) but to a target in the periphery of the field. Of course it might then be argued that the target in the left field served as a new figure, while the fixation point became part of the background—functionally speaking, the area of "best performance" might have become part of the peripheral field—yet all this is speculation.

We are unable to explain the effect of amobarbital. We have a patient (F. M.) now at Bellevue Psychiatric Hospital who shows many of the phenomena found in patient H. S. Amobarbital sodium in F. M. also produced a spread of the area in the body in which allesthesia could be demonstrated. Before the injection, the allesthesia was limited to the upper extremity and most apparent in the hands and fingers. During amobarbital intoxication the allesthesia involved the entire half of the body; when F. M. was pricked or touched on his left side, he indicated that he was stimulated at a corresponding point on the right side of the body. Incidentally, in this patient there was no motor weakness or apraxia; he was able to use both hands well.

Dr. Goldstein remarked that there are cases on record in which allesthesia was present in a patient who was mentally not confused. In our experience, every one of the patients with allesthesia had severe mental symptoms. Characteristically, there were defects in abstract thinking and an inability to perform an act which involved two or more different aspects of a situation. In our attempts to determine the possible localizing significance of allesthesia, we have had no success. Since in our cases the pathologic change in the brain was diffuse, we do not know what particular focus may have to do with the phenomenon of allesthesia.

I agree with Dr. Lauretta Bender that the problem goes far beyond the question as to what part of the brain is involved. Allesthesia must be the result of a disturbance in total brain function. At present we are not even certain whether

allesthesia is primarily a motor or a sensory disturbance. We believe that it is a disorder in which sensory and motor functions are altered in a systematic fashion, but why and how this alteration occurs is a moot point. I do not know whether it is due to a contraction in the total field or not. In our cases allesthesia was only one of many symptoms, but in all cases there were certain manifestations in common. It was a matter of how we examined the patient. The more tests we introduced, the more defects we found. Besides (1) allesthesia for one half of the body, or for the upper of lower half, all patients showed (2) the phenomenon of extinction, (3) alterations in muscle tone, (4) disturbance in body image, (5) failure of right-left discrimination and other types of disorientations in space, (6) inability to perform an act in which there were two or more different aspects to the situation and (7) general concretization in thinking. Finally, in 2 cases with severe involvement there was anosognosia. As I have said, the pathologic process is diffuse and the causation is varied. It can be degenerative, vascular, syphilitic and neoplastic.

DR. HENRY ALSOP RILEY, New York: Has postmortem examination been done in any of these cases?

DR. M. B. BENDER: One of the patients (A. W.) died three weeks ago, but we are not prepared to say anything as to what was found histologically. Grossly there was atrophy of convolutions of the frontal and, especially, of the parietal lobes on both sides.

WALSHE'S TEXTBOOK ON DISEASES OF THE NERVOUS SYSTEM

Critical Comments

ROBERT WARTENBERG, M.D.
SAN FRANCISCO

BEFORE me is a book entitled "Diseases of the Nervous System Described for Practitioners and Students," by F. M. R. Walshe, edition 5, 1947. The author is one of the leading neurologists in Great Britain, known particularly for his fundamental research on the pathophysiology of motility. As a successor to Henry Head and Gordon Holmes, he has been editor of the venerable *Brain* since 1938.

His book has become so popular that it has reached the fifth edition within six years. It has been very well received by the medical press. The reviewers praised the author's "sage attitude," his "authoritative eminence." The clarity, brevity, terseness and thoroughness of the book have been emphasized. It has been showered with adjectives such as "very excellent," "most practical and useful" and has been recommended "without reservation." One reviewer[1] said: ". . . it should be in the hands of all medical men—practitioners as well as specialists." Although deserving of such praise in general, the book, on critical analysis, reveals facets which, because they concern important questions of practical neurology, seem worthy of discussion.

On page 12 Walshe speaks of eupraxic functions of "the frontal cortex immediately anterior to the so-called 'motor cortex' in which the pyramidal system arises," and on page 52 he says: "The lesions underlying motor apraxia are situated in the frontal lobe immediately anterior to the motor cortex. . . ." To be true, Walshe speaks of motor apraxia, but he mentions the eupraxic functions in general and does not speak of apraxia due to lesions of the corpus callosum, of the supramarginal, angular gyrus or of the parietal lobe. Such oversimplification of the matter is hardly permissible, even in a text of such small dimensions.

On page 20 appears the statement:

The normal plantar reflex (flexor plantar response) is another cutaneous reflex of clinical importance. Stroking or scratching of the sole should evoke a plantar-

1. Walshe, F. M. R.: Diseases of the Nervous System, Baltimore, Williams & Wilkins Company, 1947, Book Review, J. A. M. A. **136**:806 (March 13) 1948.

flexion of the great and little toes and usually of the entire foot, with an associated contraction of tensor fascia [misprint: should be "fasciae"] femoris and of quadriceps: an accompaniment not found with an extensor plantar response.

Disregarding the associated contraction of the quadriceps on stimulation of the sole, let us consider only the occurrence of associated contraction of the tensor fasciae latae ("latae"—according to the new nomenclature). A physiologically and clinically important question is raised here: What is happening to the normal tensor fasciae latae reflex on the side of the pyramidal lesion, i. e., when the Babinski reflex is present? What Walshe says means: The reflex of the tensor fasciae latae is not found with an extensor plantar response, i. e., with a Babinski reflex. This statement is supported somewhat by the investigations of Dide and Chenais,[2] and of Sabbatini,[3] who found that in a pyramidal lesion the fasciae latae reflex is diminished or not influenced at all. On the other hand, the following witnesses testify against the contention of Walshe: Crocq [4] (Belgium), Renault,[5] Déjerine,[6] Klippel and Monier-Vinard [7] (France), Goldflam [8] (Poland), Foerster [9] (Germany), Bing [10] (Switzerland), Putnam [11] (America), Pedersen [12] (Denmark) and, last but not least, Brissaud [13] himself, who, in 1896, first described this reflex. Renault [5] came to no definite conclusion about the behavior of the reflex of the tensor fasciae latae in organic hemiplegia but found it preserved or exaggerated in compression of the spinal cord above the fourth lumbar segment, while the plantar reflex was lost. Déjerine [6] said: "When the Babinski reflex exists one finds that the tensor

2. Dide, M., and Chenais, L.: Sur le réflexe du fascia lata, J. de neurol. et de psychiat. **7**:275, 1902.

3. Sabbatini, G.: Contributo allo studio del riflesso del tensore della fascia lata, Riv. ital. di neuropat. **5**:97, 1912.

4. Crocq, J.: Le réflexe du fascia lata, J. de neurol. et de psychiat. **7**:28, 1902.

5. Renault, E.: Etude du réflexe du fascia lata, Thesis, Paris, no. 268, 1903.

6. Déjerine, J.: Semiologie des affections du système nerveux, ed. 3, Paris, Masson & Cie, 1927, p. 956.

7. Klippel, M., and Monier-Vinard, R., in Roger, G. H.; Widal, F., and Teissier, P. J.: Nouveau traité de médecine, Paris, Masson & Cie, 1925, vol. 19, p. 11.

8. Goldflam, S.: Zur Lehre von den Hautreflexen an den Unterextremitäten (insbesondere des Babinski'schen Reflexes), Neurol. Centralbl. **22**:1109, 1903.

9. Foerster, O.: Die Erkrankungen der peripheren Nerven, in von Schjerning, O.: Handbuch der ärztlichen Erfahrungen im Weltkriege 1914-1918, Leipzig, J. A. Barth, 1922, vol. 4, pt. 1, p. 269.

10. Bing, R.: Lehrbuch der Nervenkrankheiten, ed. 8, Basel, Benno Schwabe, 1947, p. 171.

11. Putnam, T. J.: Neurologic Examination in General Practice, M. Clin. North America **22**:555, 1938.

12. Pedersen, E.: On the Tensor Fasciae Latae Reflex, Acta psychiat. et neurol. **23**:113, 1948.

13. Brissaud, E.: Le réflexe du fascia lata, Gaz. hebd. de. méd. **1**:253, 1896.

fasciae latae reflex behaves as in normal conditions and does not suffer any modification." Klippel and Monier-Vinard [7] said that this reflex is exaggerated in hemiplegia, in which it is associated with the Babinski sign. Foerster [9] mentioned that "on stroking the foot the reflex contraction of the tensor fasciae latae is much more extensive in spastic paralysis of the leg than in normal." Bing,[10] describing the Babinski phenomenon said that "the dorsiflexion of the toe can be associated with other motor phenomena as . . . simultaneous reflex twitchings in the tensor fasciae latae, . . . in the quadriceps." All other workers mentioned have expressed similar views, particularly Pedersen [12] in his review which appeared in 1948. Putnam even defined "a positive (abnormal) response" on stimulation of the sole of the foot as "slight dorsiflexion of the great toe, a spreading of the other toes and a contraction of the tensor fasciae latae." Brissaud,[13] in his original description of this reflex, reported a case of syringomyelia, in which, on the paretic side, this reflex coincided with the Babinski reflex. My own experience gives me no reason to doubt that the tensor fasciae latae reflex is not only present in spastic paralysis of the leg but is nearly always exaggerated, and often very markedly so. Physiologically this is easily understandable, since the tensor fasciae latae muscle participates in the mass flexion movement of the lower extremity, occurring when the sole is stroked. However, the tensor fasciae latae reflex is really lost in lesions of the lower motor neuron, as for instance in "radicular sciatica." This has been reported by Bonola.[14]

In figure 3 (opposite page 22), which originally appeared in a former work of Walshe,[15] a very important phase of the Babinski reflex is graphically illustrated. On stimulation of the left toe in spastic paraplegia, there is a homolateral mass flexion reflex with dorsiflexion of the great toe and of the foot, whereas on the contralateral side there is a mass extension reflex with plantar flexion of the toe and of the foot. Unfortunately, everywhere in his book, Walshe speaks of the Babinski reflex as an "extensor plantar response," though this response is only part of a mass flexion movement of the lower limb. Thus it has happened that the legend accompanying this illustration reads as follows: "The Extensor and Crossed Extensor Plantar Responses." This means that the dorsiflexion of the toe on the left side is called an extensor response, and the plantar flexion of the toe on the right side is also called an extensor plantar response. This is highly confusing! It would have been better, of course, to speak of a "homolateral

14. Bonola: Les réflexes du tendon d'achille et du tenseur du fascia lata, dans la sciatique radiculaire et dans la sciatique tronculaire, Rev. neurol. **24**:324, 1912.

15. Walshe, F. M. R.: The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, Brain **37**:269, 1914 (fig. 6).

mass flexion reflex" and of a "contralateral mass extension reflex" and to refer to the Babinski reflex as a "dorsiflexion of the toe."

Discussing the symptomatology of cerebellar lesions, Walshe says (p. 30):

> . . . if the patient be asked to perform alternating movements, such as alternate pronation-supination of the wrist, this inherent defect—intention tremor—becomes aggravated. The movements are irregular in rate and amplitude—generally excessive in range—and are thus slower than normal.

According to this, when the patient is tested for adiadokokinesis "the intention tremor becomes aggravated." Mind, that Walshe does not say "the intention tremor appears"; he says "becomes aggravated." This would appear to presuppose a rest tremor in cerebellar disease which is, of course, untenable. My own inadequacies may be blamed exclusively, but I must confess that I would be completely at a loss to know how to explain to students that it is the intention tremor which becomes aggravated in adiadokokinesis. The clinical manifestations of the two conditions obviously differ too widely. The cerebellar speech, for instance, is most probably due to adiadokokinesis. But who would venture to speak here of "intention tremor becoming aggravated"? It is not the intention tremor, but dyssynergia, that should be regarded as a fundamental disturbance in cerebellar disease, as has been shown by Ramsay Hunt.

Of the rib pressure syndromes Walshe says (p. 234): "Tenderness on pressure is, however, virtually constant in opponens pollicis and less frequently in the first dorsal interosseous muscle." In sciatica, "all muscles supplied by the nerve are extremely tender when pressed or stretched, and they become flabby" (p. 274). Still more apodictic and all-embracing is the following statement: "The degenerating muscle wholly, or even partly, cut off from the nervous system by a lesion of the lower motor neurone is usually extremely tender to pressure, . . ." (p. 31). Among neurologists anywhere, there is scarcely one whose experience would permit him to subscribe to these statements as they stand.

It would have been better to omit the 26 lines devoted to electrical examination (p. 31). One has the definite impression that the author is not at home in this field. He says, for instance, ". . . that it is comparatively rare for this testing to provide diagnostic information that cannot more easily and reliably be obtained by other means." Though it is a matter of personal opinion, I believe I am correct in stating that most authorities would disagree with Walshe on this point. Walshe further writes: "Healthy muscle in normal connection with the nervous system responds by a tetanic contraction when its motor nerve is stimulated by a faradic current. The muscle itself

does not respond to this form of stimulation, but only to the continuous or galvanic current. It then does so by a short-lived contraction when the current is made and broken." All this is not clear, to say the least. Which muscle ". . . does not respond?" The diseased or the healthy one? A "short-lived contraction" occurs in both. It is the slow wave which is characteristic of the diseased muscle. The sentence as it stands may be interpreted by the student to mean that the normal muscle itself does not respond to the faradic current. Certainly Walshe did not want to convey this impression.

On page 37, Walshe speaks of astereognosis. However, from the context one is forced to assume that he wishes to speak of disturbances in the peripheral sensory pathways. In such a case, one should speak not of astereognosis but of stereoanesthesia and reserve the term astereognosis for disturbances of higher cortical centers. One does not say "visual agnosia" if the optic nerve is damaged. "One must be cautious," says Nielsen,[16] "not to ascribe to astereognosis defects of identification due to lesions of peripheral nerves such as occur in pernicious anemia or polyneuritis of any sensory form, or due to anesthesia from a lesion of the sensory tract or of the thalamus."

On page 65 in the discussion of headaches in hysteria and in anxiety neurosis Walshe says: "In both circumstances, the eager desire to impress at all costs . . . are evident." No! This "desire to impress at all costs" is true only for the hysterical patient and by no means for those with anxiety neuroses in general.

The following sign is, according to Walshe, (p. 89) "usually easy of elicitation" in the case of a cerebellar abscess: "If the arms be horizontally extended the arm on the side of the abscess tends to droop." Such drooping of the horizontal arm, it is true, may occur on the side of a cerebellar affection. It occurs in other affections as well, however, and since it is by no means specific for cerebellar dysfunction, it cannot be considered a proper cerebellar sign. On the contrary, on the side of a cerebellar lesion one may see what is called a "spontaneous rise reaction," and a "spontaneous divergence reaction." These reactions, which exist in mild form in the normal, tend to be exaggerated on the side of the cerebellar lesion. When the patient—with, for instance, a right-sided cerebellar affection—holds his horizontally outstretched arms in front of him, the right arm has a tendency to rise and to deviate laterally. In their monograph, Hoff and Schilder,[17] discussing this "spontaneous rise reaction," illustrate it graphically (their fig. 13). The

16. Nielsen, J. M.: A Textbook of Clinical Neurology, ed. 2, New York, Paul B. Hoeber, Inc., 1946, p. 245.

17. Hoff, H., and Schilder, P.: Die Lagereflexe des Menschen. Vienna, Julius Springer, 1927, p. 82.

patient with a cerebellar tumor on the left side involuntarily raises his left arm and moves it laterally when his arms are outstretched. The tendency for the homolateral arm to rise in cerebellar affection is also well illustrated by the "imitation phenomenon," which is closely related to the "spontaneous rise reaction." When, in a patient with right-sided cerebellar affection, the examiner puts the patient's left arm in a certain position and asks him to imitate this with his right arm, the patient involuntarily and unconsciously raises the right arm higher than the left. It is obvious, then, that drooping of one of the horizontally extended arms can hardly serve as a useful test for homolateral cerebellar dysfunction.

In discussing epilepsy, Walshe says (p. 121) that postepileptic automatism occurs after minor attacks. It is not quite correct to speak of minor attacks only, since epileptic automatism may occur after minor and major attacks or before them. It may also occur in place of a major or minor attack. It is true, however, that postepileptic automatism occurs much more frequently than preepileptic. It was formerly assumed that psychomotor attacks in epilepsy did not occur unless preceded by attacks of grand mal or petit mal, but this theory has been abandoned. Bleuler [18] said in his textbook that "twilight states" most frequently occur postepileptically but may also take place preepileptically and instead of the attack ("equivalents").

Walshe reports (p. 129) on attempts made to give larger doses of phenobarbital in epilepsy, the narcotic effects being counteracted by the additional administration of amphetamine sulfate (benzedrine sulfate®), 15 to 30 mg. daily, but fails to clarify his own position regarding such treatment. The administration of the drug (15 to 30 mg. daily) would necessarily be extended over a long period of time, perhaps indefinitely. Against this I would raise a most serious objection. Though occasionally a patient can tolerate amazingly large amounts of amphetamine, it is to be feared that given in such high doses for long periods of time it might produce untoward, disastrous results in many systems. At least, in the face of such heroic treatment, the slow increase of dosage and the closest observation is imperative, and this point should be most emphatically stressed. It is not.

On page 180, in the chapter on multiple sclerosis, the reader is startled by the following statement: "We know of no virus that acts upon white matter. . . ." However, to my knowledge, there are many demyelinating diseases, such as disseminated encephalomyelitis caused by exanthems, leukoencephalitis, herpetic meningoencephalitis, cowpox encephalitis, St. Louis encephalitis, neurotropic myelitis and many other morbid conditions of the nervous system, which may be attributed to

18. Bleuler, E.: Textbook of Psychiatry, New York, The Macmillan Company, 1924, p. 453.

viruses acting on the white matter. The statement of Walshe appeared to me to be incorrect, to say the least. But, being only a medical neurologist, I inquired of several leading neuropathologists in the United States, asking their opinion on Walshe's statement. The answers were unanimous, varying only quantitatively from "not true" and "decidedly wrong" to "false."

In the treatment of paralysis agitans (p. 193) it is stated: "For those patients who are restless or sleepless small regular doses of phenobarbitone (gr. ¼ to ½ twice or thrice daily) are more useful than the bromides. If this be not adequate to deal with insomnia, there should be no hesitation in giving larger doses of a barbiturate." This brings to mind that Ziskind and Ziskind [19] in 1937 found that phenobarbital aggravates rigidity in parkinsonism. They concluded that "phenobarbital, and probably the other barbiturates as well, are contraindicated in parkinsonism." In view of this statement of experienced neurologists, I must confess I should certainly hesitate—and thoughtfully—before advising the administration of "larger doses of a barbiturate" in paralysis agitans. Ziskind, in a private communication (1948), verified his experience in 13 consecutive cases, whereas the original report was based on but 5 cases. He learned of other physicians who had had similar experiences. He considers the unfavorable reaction of patients with parkinsonism to phenobarbital as a definitely established fact.

"Within the theca we may find diffuse sarcomatosis, and small neurofibromata, more often single than multiple, may grow from the sheaths of spinal nerve roots" (p. 205). Exactly the opposite is true: Small neurofibromas inside the theca are found more frequently multiple than single. In his own book, figure 59 shows "very numerous neurofibromata on the surface of the cord and studding the roots of the cauda."

"Haemorrhage plays no significant role in spinal cord injuries, and what has been called haematomyelia is not simply a result of injury, but an extremely rare lesion associated with some form of arterial abnormality" (p. 206). From what may be learned on the problem of hematomyelia as set forth in the international literature, it would appear that such a drastic and conclusive statement is by no means warranted. It is true that spontaneous hematomyelia may result from intramedullary angioma or syphilis of the spinal cord (Richardson [20]), or from hemangioma (Buckley [21]). But this gives one no reason to deny the

19. Ziskind, E., and Ziskind, E. S.: Phenobarbital Contraindicated in Parkinsonism, J. A. M. A. **109**:20 (July 3) 1937.

20. Richardson, J. C.: Spontaneous Haematomyelia: A Short Review and a Report of Cases Illustrating Intramedullary Angioma and Syphilis of the Spinal Cord as Possible Causes, Brain **61**:17, 1938.

21. Buckley, A. C.: Hematomyelia Secondary to Hemangioma, J. Nerv. & Ment. Dis. **83**:422 (April) 1936.

existence of a traumatic hematomyelia in which there is no indication of a premorbid affection of the spinal arteries. Here reference might be made to hematomyelia occurring in athletes, to hematomyelia of divers (Guillain,[22] Picard[23]). A case observed by me and reported by Cohn[24] leaves no doubt of the purely traumatic origin of the affection. The fact that hematomyelia—especially that which affects the lower part of the spinal cord—can heal completely, or almost completely, without any sequelae speaks in favor of the assumption that there is a hematomyelia which is purely traumatic in origin and for which it seems unnecessary to assume a preexisting arterial abnormality. The well established, time-honored term "traumatic hematomyelia" still holds. Bing[25] used the term "traumatic hematomyelia" and listed a considerable number of conditions under which it occurs.

The occurrence of muscular contracture in myopathy is unduly stressed. On page 222 Walshe says: "In the final stage the muscles undergo a fibrous change, shorten, and thus lead to the development of skeletal deformities and contractures of the limbs." On page 224 he speaks of ". . . the inevitable process of muscular contracture." To this one could say that muscular contracture occurs in dystrophy not only very late but very rarely! Several textbooks on neurology even fail to mention muscular contractures in myopathy. Bing[26] says, "that atrophic or pseudohypertrophic muscles shorten, and thus lead to contractures . . . is not a common occurrence." If muscular contracture had been "the inevitable process" in muscular dystrophy, Cestan and Lejonne,[27] in 1902, would not have called attention to 2 cases of myopathy with contractures which they considered extraordinary and spoken of "abnormal deformities." Nor would Steinert[28] in 1910 have separated a special form called "dystrophia musculorum progressiva retrahens"! Still more striking are the results Walshe attributes to contractures in muscular dystrophy. On page 223, he says: "The flexors of the thigh are not readily visible, but they also are wasted and shortened *so* [italics mine] that full extension of the thigh is impossible. *Hence* [italics mine] when standing there is a marked lordosis." The italicized "so" and "hence" are significant here.

22. Guillain, G.: Le danger des plongées par fond d'eau insuffisant, l'hématomyélie des plongeurs, Bull. Acad. de méd., Paris **105**:996, 1931.

23. Picard, H.: Ueber einen Fall von Hämatomyelie bei Kopfsprung, Thesis, Basel, 1937.

24. Cohn, E.: Zur Klinik der posttraumatischen Hämatomyelie mit Intervall, Arch. f. Psychiat. **95**:439, 1931.

25. Bing,[10] p. 229.

26. Bing,[10] p. 153.

27. Cestan, R., and Lejonne, P.: Une myopathie avec rétractions familiales, Nouv. iconog. de la Salpêtrière **15**:38, 1902.

28. Steinert, H.: Myopathologische Beiträge, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **21**:105, 1910.

They mean that, according to Walshe, the explanation for the lordosis in dystrophy is as follows: The flexors of the thigh are wasted and shortened and prevent full extension; hence the lordosis. This is not correct. Lordosis in dystrophy always appears so early in both young and old that it cannot be explained by muscular contractures, which are late, rare and—oftener than not—absent. I have never seen lordosis in dystrophy attributable to contracture. Such lordosis is due to weakness of the muscles that stretch the hip joint and not to the contracture of the flexors of the thigh.

On page 228 appears the statement, ". . . most cases so labelled [primary lateral sclerosis] ultimately prove to be amyotrophic lateral sclerosis or some other known affection of the nervous system, and the title corresponds to no special recognized pathological process and should be abandoned." According to Walshe himself, "most" cases of primary lateral sclerosis eventually prove to be something else—"most", but not all, of them. For this remainder, whatever its size, the term "primary lateral sclerosis" should be reserved. There is no sound reason to abandon it completely. Since Erb [29] in 1877 established the syndrome of spastic spinal paralysis, attempts have been made to detract from the sovereignty of the syndrome and to prove its secondary character. Despite impingement from all sides, the syndrome of primary lateral sclerosis still stands, both clinically and pathologically. Suffice it to mention the work of Strümpell,[30] who observed the patient named Polster for fifteen years and said: "I have never had the slightest doubt about the diagnosis of the primary lateral or pyramidal sclerosis. The postmortem examination has proved this diagnosis to be absolutely correct." Stark and Moersch [31] in 1945 said: ". . . we are justified in stating that in a limited number of cases a diagnosis of primary lateral sclerosis seems permissible." Wechsler and Brody [32] in 1946 said: "Primary lateral sclerosis seems to be a distinct clinical syndrome unrelated to other scleroses." Riley [33] mentioned that lateral sclerosis "may be a primary disease." "But cases still remain," said Bing,[34] "which prove themselves as genuine systemic degeneration of the

29. Erb, W.: Ueber die spastische Spinalparalyse (Tabes dorsal spasmodique, Charcot), Virchows Arch. f. path. Anat. **70**:241, 1877.

30. Strümpell, A.: Die primäre Seitenstrangsklerose (spastiche Spinalparalyse), Deutsche Ztschr. f. Nervenh. **27**:291, 1904.

31. Stark, F. M., and Moersch, F. P.: Primary Lateral Sclerosis: A Distinct Clinical Entity, J. Nerv. & Ment. Dis. **102**:332 (Oct.) 1945.

32. Wechsler, I. S., and Brody, S.: The Problem of Primary Lateral Sclerosis, J. A. M. A. **130**:1195 (April 27) 1946.

33. Riley, H. A., in discussion on Wechsler and Brody,[32] p. 1198.

34. Bing,[10] p. 167.

cortico-spinal tracts in the area of their spinal course." In 1946 Gordon [35] reported two new "pure or unmixed cases" of primary lateral sclerosis which he had "observed uninterruptedly" over a period of twenty-four years. There has never been any indication that here the lesion extended to portions of the white matter of the spinal cord other than that of the pyramidal tract. It is a matter of common clinical experience that the syndrome of lateral sclerosis may usher in quite a number of diseases of the spinal cord. That many cases may be of this type, however, will in no way negate the fact that the syndrome of primary lateral sclerosis does exist. In over thirty years' experience in Europe and the United States, I have seen cases of primary lateral sclerosis which have remained "primary," even after more than ten years' observation. I can only agree fully with Stark and Moersch,[31] who spoke of primary lateral sclerosis as "a distinct clinical entity." Thus it would appear that no valid reason exists for proposing that the name of primary lateral sclerosis "be abandoned."

In the section entitled "Pressure Symptoms Produced by a Normal First Rib, and the So-Called Scalenus Anticus Syndrome," Walshe mentions (p. 236) "that relief of symptoms is sometimes achieved by section of the tendon of this muscle [scalenus anticus] . . .," but under "treatment" (p. 237), he says "surgical removal of the rib . . . is necessary." Such heroic advice will startle every neurosurgeon. Referring to his operation, Adson [36] said (1928): "The anterior approach and tenotomy of the scalenus anticus are preferable to the transcervical approach and resection of the cervical rib since the same relief is offered, the procedure is less formidable. . . ." In 1945 Adson [37] said: ". . . division of the tendinous insertion of the scalenus anticus muscle is the only procedure employed. This same surgical procedure is employed for the relief of the scalenus anticus syndrome, a group of symptoms simulating those produced by a cervical rib." In an article in 1947, Adson,[38] speaking of twenty-two years' experience in the treatment of cervical ribs and of the scalenus anticus syndrome, said: "Section of the scalenus anticus muscle which I suggested and reported on in 1927, instead of removal of the supernumerary rib, continues to be the operation of choice."

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35. Gordon, A.: The Clinical Status of Unmixed Types of Primary Lateral Sclerosis After Twenty-Four Years of Observation, J. Nerv. & Ment. Dis. **103**: 378 (April) 1946.

36. Adson, A. W.: Cervical Rib, Atlantic M. J. **31**:222 (Jan.) 1928.

37. Adson, A. W.: Cervical Rib, in Christopher, F. A.: Text Book of Surgery, ed. 4, Philadelphia, W. B. Saunders Company, 1945, p. 412.

38. Adson, A. W.: Surgical Treatment for Symptoms Produced by Cervical Ribs and the Scalenus Anticus Muscle, Surg., Gynec. & Obst. **85**:687 (Dec.) 1947.

On page 237 it is said: "The subjects [with acroparesthesia] are debilitated, tired and overworked women. . . ." Such generalization is by no means justifiable. It would be much better to say: "Often the subjects. . . ."

"The so-called chronic retrobulbar neuritis is in reality a toxic affection of the ganglion cells of the retina caused by various poisons—e. g., quinine, methyl alcohol, tobacco ('tobacco amblyopia') . . ." (p. 258). It is true, the time-honored theory that the intoxication amblyopias are caused only by changes in the optic nerve has undergone criticism from several sides. The changes in the ganglion cells in the retina have been stressed. Despite this, what is now known on this score by no means permits one to throw the old theory overboard. It would be decidedly premature. I quote the ophthalmologist, Marchesani [39]: "Opinions differ as to whether the degeneration attacks the optic nerve or the retina."

On page 261 one learns that in trigeminal neuralgia the pain makes its first appearance "never in the first division." This "never" is simply not correct. No less a man than Romberg,[40] speaking of facial neuralgia, said in 1853: "One of my patients had for eight years an annual autumnal attack of frontal neuralgia, with a quotidian type." In his classic treatise on neuralgia Anstie [41] wrote of trigeminal neuralgia: "The most frequent occurrence is the limitation of the pain to the ophthalmic division. . . ." Harris [42] wrote: "It is very rare for the pain to start in the first division alone, but occasionally this sequence is noticed, the pain being limited to the first division sometimes for as long as three or four years before it begins to invade the second division." Such an experienced neurologist as Bing [43] stated that trigeminal neuralgia "may begin in one of the divisions and subsequently spread to adjacent areas." Wexberg,[44] too, stated that he has seen cases of primary genuine neuralgia of the first branch, though rarely. Davis and Naffziger [45]

39. Marchesani, O.: Symptomatologie des Nervus opticus (einschliesslich Stauungspapille), in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 90.

40. Romberg, M. H.: A Manual of the Nervous Diseases of Man, London, The Sydenham Society, 1853, vol. 1, p. 34.

41. Anstie, F. E.: Neuralgia and the Diseases that Resemble It, New York, Bermingham & Company, 1882, p. 28.

42. Harris, W.: The Facial Neuralgias, London, Oxford University Press, 1937, p. 19.

43. Bing, R., and Haymaker, W.: Textbook of Nervous Diseases, St. Louis, C. V. Mosby Company, 1939, p. 107.

44. Wexberg, E.: Neuralgien, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1935, vol. 9, p. 199.

45. Davis, E. W., and Naffziger, H. C.: Major Trigeminal Neuralgia: An Analysis of Two Hundred and Forty-Five Cases, California Med. **68**:130 (March) 1948.

commented that the onset of pain in the first division is "quite rare." Hughes [46] said of trigeminal neuralgia: "On the other hand, but more rarely, the attacks may originate in and remain confined to the first division." Coleman, Meredith and Troland [47] saw 4 patients with tic douloureux, with involvement of the first division alone. In view of these authoritative statements, made between 1853 and 1948 the word "never," as used by Walshe, is not justifiable.

Speaking of electrical stimulation of the paralyzed muscles of the facial nerve, Walshe says (p. 265): "Indeed, if persisted in it may lead to disfiguring contracture of the partly recovered muscles." Again on page 303 ". . . in slowly recovering cases and especially in elderly persons the galvanic stimulation of the muscles leads to distressing contracture and should on no account be employed." I quote this statement without comment. Every neurologist who remembers his own cases of contractures occurring in untreated and partially recovered facial palsy, who takes into consideration the pathophysiology of such contractures and accompanying associated movements (Wartenberg [48]) will be amazed to find such views expressed in a work published A. D. 1947. Walshe referred to himself (p. 197) as "old-fashioned," but even such an admission is not an extenuating circumstance.

"Lipiodol intrathecally, formerly used . . ., has been abandoned." (p. 275). It has not—to the best of my knowledge, at least.

In describing lesions of the upper brachial plexus, involvement of the musculus subscapularis is mentioned on page 281 (but not on page 296). I doubt that this muscle is involved in upper brachial plexus palsy.

Speaking of meralgia paresthetica (p. 289), Walshe says: "It occurs in males." This reminds me that in the cases of Fischer and Krieg [49] meralgia paresthetica affected women predominantly. Generally speaking, however, it is found more commonly in the male than in the female, according to statistics published by various authors in the following ratios respectively: 3:2, 2:1, 4:1, 12:2, 25:7. One compilation in the literature lists 88 cases of its occurrence in the male and 24 in the female. Since meralgia paresthetica does occur in the female, there is no reason for Walshe's terse and summary statement.

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46. Hughes, B.: The Radical Treatment of Trigeminal Neuralgia, Brit. M. J. **1**:823 (May 8 and 15) 1926.

47. Coleman, C. C.; Meredith, J. M., and Troland, C. E: Tic douloureux: Review of Two Hundred Cases Treated by Surgery, Virginia M. Monthly **75**:386 (Aug.) 1948.

48. Wartenberg, R.: Associated Movements in the Oculomotor and Facial Muscles, Arch. Neurol. & Psychiat. **55**:439 (May) 1946.

49. Fischer, E., and Krieg, E.: Zur Symptomatologie und Therapie der Meralgia paraesthetica, München. med. Wchnschr. **79**:710 (April 29) 1932.

It seems doubtful to me that in paralysis of the lower brachial plexus the limb is held abducted (p. 297). Walshe stands alone when, on page 341, he assigns the cremasteric reflex to the twelfth thoracic segment. It is usually assigned to the first, second and even third lumbar segments. Loewy [50] concluded his detailed treatise on the cremasteric reflex: "The reflex center of the cremasteric reflex lies in the first to third lumbar segment."

Walshe often uses the term "abdominal reflexes." Such a term should not be used, since distinction must be made between the abdominal muscle reflexes and the abdominal skin (superficial) reflexes. In a pyramidal lesion the former are exaggerated, the latter lost. To say "abdominal reflexes" and mean "abdominal skin reflexes" is confusing.

It has always been tacitly understood that there is a differentiation between "signs" and "symptoms." Gordon Holmes [51] rightly stated: "A distinction is usually made between symptoms, that is, disorders described by the patient, and physical signs or the deviations from the normal revealed by examination and though such a distinction cannot be absolute . . . it is useful in clinical investigations." Walshe himself speaks (p. 11) of "subjective symptoms and objective signs," but one is surprised to find on page 235 the statement, "The symptoms in these cases are commonly largely subjective. . . ." He designates such phenomena as tremor, rigidity, athetosis, facial hemispasm (p. 6), all the release phenomena due to a lesion of the upper motor neuron, including the Babinski reflex (p. 21), phenomena due to a lesion of the extrapyramidal system (p. 27) as symptoms. Slowing of the pulse rate (p. 68), squint, diplopia, edema of the optic disk, retinal hemorrhages, etc. (p. 113) are called symptoms. Again, nystagmus, dysarthria (p. 218) and bruit over the subclavian artery (fig. 46) are similarly classified.

The deep muscle reflexes are sometimes called "reflexes," sometimes "jerks." The term "tendon reflexes" is just as obsolete as the term musculus "supinator longus" for "brachioradialis."

The test described by Romberg is usually called the Romberg test or simply "Romberg." Walshe uses the abhorrent term "Rombergism." It is hoped that no one will follow him. This would only lead to such barbarisms as "Baranyism," "Queckenstedtism," "Naffzigerism"!

Walshe's style is not always smooth. Involved sentences and such phrases as "occasional case" (pp. 211, 228, 230) and . . . "quiet are required" (p. 197) contribute to this end.

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50. Loewy, E.: Beitrag zum Verhalten des Kremasterreflexes bei funktionellen und organischen Nervenkrankheiten inkl. Psychosen, Monatschr. f. Psychiat. u. Neurol. **32**:525, 1912.

51. Holmes, G.: Introduction to Clinical Neurology, Baltimore, Williams & Wilkins Company, 1946, p. 1.

There are a few misprints that should not occur in the fifth edition of a textbook. For instance: fascia for fasciae (p. 20); check for cheek (p. 24); sigh for sign (p. 47); about for a bout (p. 123); facia for facial (p. 262); spiral for spinal (p. 305); Brown-Séquard appears without *accent aigu* (p. 38, 39).

But withal it is an interesting, stimulating and readable book and brings to mind what Oliver Goldsmith said about his Vicar of Wakefield: "A book may be amusing with numerous errors, or it may be very dull without a single absurdity."

University of California Hospital (22).

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Anatomy and Embryology

DEVELOPMENTAL DEFECTS OF THE CISTERNA MAGNA AND DURA MATER. E. G. ROBERTSON, J. Neurol., Neurosurg. & Psychiat. **12**:39 (Feb.) 1949.

Robertson records a number of variations in the normal arrangement of the cisterna magna and emphasizes their significance in failures in ventricular filling during encephalographic procedures. The lateral limits of the cistern may be unduly wide and are placed at a higher level than the foramen of Magendie; thus, gas entering dorsally will tend to flow forward at a higher level, to the cerebellopontile angle and into the pontile cisterns. Another defect is failure of the pia and arachnoid to fuse over the inferior surface of the cerebellum, eliminating the usual barrier at the upper end of the cisterna magna; thus, gas can pass around the cerebellum to the cisterna venae magnae cerebri and the cerebral subarachnoid space, not being directed to the foramen of Magendie. In other anomalies the cisterna cerebellomedullaris (magna) is observed to extend as high as the internal occipital protuberance. The author found 4 instances of this anomaly in 100 cases. In the fourth case autopsy showed a globular projection of dura mater between the occipital poles, arising through a hiatus in the tentorial shelf. Apparently, the posterior inferior portion of the falx cerebri had split into two layers, which laterally became continuous with the tentorium. The anterior part of the falx cerebri was also malformed, so that there was no dura between the medial surfaces of the frontal lobes. A developmental lesion seemed the most likely explanation. In this case there was a coincident cyst in the vermis, which appeared on microscopic examination to be of ependymal origin.

N. MALAMUD, San Francisco.

ABNORMAL COURSE OF THE FACIAL NERVE IN THE FALLOPIAN CANAL. KARSTEN KETTEL, Arch. Otolaryng. **44**:406 (Oct.) 1946.

Discoveries drawn from observations in 125 operations on the facial nerve performed in the manner of Ballance and Duel are presented as controversial evidence against the rule stated in textbooks of anatomy and otology to the effect that the facial nerve is never found dorsal to the frontal plane through the tympanomastoid suture. Bridgett and Batson, as a result of their observations, attributed a normal course to the facial nerve in all cases. However, Hawley reported 4 instances in 300 dissections made in which the nerve was displaced 3 or 4 mm. posterior to the suture. The author is in agreement with Hawley's findings. He substantiates his claim by describing three different ways in which

the facial nerve has exhibited variations in its course. Failure to consider the surgical significance of possible deviations of the facial nerve from its normal course will inevitably result in injury to the nerve during its decompression.

RYAN, Philadelphia.

Physiology and Biochemistry

IPSILATERAL SENSORY LOSS FOLLOWING CORDOTOMY. LYLE A. FRENCH and WILLIAM T. PEYTON, J. Neurosurg. **4**:403 (July) 1948.

French and Peyton report a case in which section of the right spinothalamic tract was followed by loss of pain and temperature on the same side. The patient, aged 59, had severe, intractable pain in the left inguinal region and the left leg, resulting from a metastatic carcinoma. A cordotomy was performed on the right side at the third thoracic segment. There was immediate loss of pain and temperature sensations from the sixth thoracic level down, but on the same side as the section. The pain continued. A cordotomy was then performed on the left side at the fifth thoracic level, with complete relief of the pain on the left side.

This anomaly could have been due to a modified cord diplegia. This anomaly has been described, but is usually in the lumbar or the low thoracic region. Autopsy in this case was not permitted.

TOZER, Philadelphia.

CURARE IN SPASTIC PARAPLEGIA. IRVING S. COOPER and THOMAS I. HOEN, J. Neurosurg. **5**:464 (Sept.) 1948.

Cooper and Hoen report the effects of curare on 8 paraplegic patients. All had severe flexor spasticity of the lower extremities. The patients were given purified chondodendion tomentosum (intocostrin®) in doses sufficient to produce side effects that made it necessary to discontinue the drug. None of the patients demonstrated a decrease in the spasticity of the affected muscles.

Cystometrograms were obtained daily during the administration of the curare. The hypertonicity of the bladder was not affected.

TOZER, Philadelphia.

Neuropathology

REACTIONS OF CEREBRAL TISSUE TO SILVER, TANTALUM AND ZIRCONIUM. J. I. BATES, F. H. LEWEY and C. R. REINERS, J. Neurosurg. **4**:349 (July) 1948.

The authors report the results of the implantation of strips of silver, tantalum and zirconium on the cerebral cortex. Silver, the most commonly used metal in neurosurgery, produced the greatest reaction. A definite fibrous capsule surrounded the implant, with necrosis and infiltration of the cortex and meninges. Zirconium produced a minimal reaction with no gross necrosis or capsule formation. Microscopically, there was a delicate connective tissue capsule, with minor marginal necrosis in the later stages. Tantalum, like zirconium, gave minimal changes. Both gross and microscopic examination of the area around the implant

showed little or no change in glial or mesodermal elements. From this study the authors recommend the use of tantalum or zirconium in cerebral operations. They advise against the use of silver.

TOZER, Philadelphia.

Psychiatry and Psychopathology

THE HYPERVENTILATION SYNDROME. WALTER FREEMAN, Dis. Nervous System **9**:180 (June) 1948.

Freeman equates hyperventilation and anxiety, stating that they are so closely related that it is impossible to treat one without the other. Hyperventilation is a common response to emotional stress and produces neurophysiologic changes through a decrease in the carbon dioxide tension in the blood, establishing the multifold symptoms of the syndrome. In treatment of anxiety states associated with hyperventilation, the author first performs a Libman test, to determine the pain threshold, and a hyperventilation test, to demonstrate to the patient the cause of his symptoms. When the result of the hyperventilation procedure is negative and the pain tolerance is high, it is felt that the patient is not suffering from an anxiety tension state but has an endogenous depression or organic disease. Treatment consists in explanation in simple terms, using the analogy of the gas mixture supplying an internal combustion engine, followed by a regimen of physical exercise, motivated by constant encouragement. Sedatives are used, in moderation, since they have the helpful effect of reducing the tendency to overbreathe. Ammonium chloride and an acid-forming diet, including some dilute hydrochloric acid, counteract to some extent the results of hyperventilation on the hydrogen ion concentration of the blood. Many patients can pull themselves out of the ruts of their illness on such a program; the demonstration of the hyperventilation test quiets fears of physical disease, and the control of the symptoms of hyperventilation relieves anxiety. Sometimes a few electroshock treatments are needed to launch the career of outdoor activity, while in those who are amenable psychotherapy is indicated for assault on the basic anxiety.

BEATON, Tucson, Ariz.

THE INTELLIGENCE OF PATIENTS WITH FRIEDREICH'S ATAXIA. D. L. DAVIES, J. Neurol., Neurosurg. & Psychiat. **12**:34 (Feb.) 1949.

Davies investigated the intellectual status in 20 patients with Friedreich's ataxia (hereditary sclerosis, spinal form). Three routine tests were used: (1) the Mill Hill vocabulary test, (2) Raven's progressive matrices and (3) the verbal half of the Wechsler-Bellevue test. None of the patients showed idiocy, imbecility or even a degree of mental defect significant to justify that term. In some patients there was a mild degree of loss of recent memory and of concentration and attention. The conceptual quotient of the patients with the disease was significantly lower than that for a control group of 15 chronic invalids, an observation suggesting that the lower rating was not accounted for merely by invalidism. The authors conclude that patients with Friedreich's ataxia are not intellectually different from the general population but, as a group, show some dementia, of no great degree.

N. MALAMUD, San Francisco.

Diseases of the Spinal Cord

TONSILLECTOMY AND POLIOMYELITIS. DANIEL S. CUNNING, Arch. Otolaryng. **46**:575 (Nov.) 1947.

The suggestion was made at the May 1946 meeting of the American Laryngological, Rhinological and Otological Society that a nationwide survey be conducted to determine the relation of tonsillectomy to poliomyelitis. Members of the society in each state were appointed to gather pertinent information for a statistical report. Some made no reports; others made reports so sketchy and incomplete as to be useless. Almost 4,000 of the 6,000 questionnaires returned had to be discarded. Although the study cannot settle conclusively the problem whether surgeons should avoid removing tonsils during epidemics of poliomyelitis, some valuable evidence was obtained. For instance, in 4.7 per cent of the cases of bulbar involvement the illness was preceded by tonsillectomy, while in only 1.9 per cent of the cases of spinal involvement did the poliomyelitis follow recent tonsillectomy. Even these values must be considered inconclusive because of inadequate data and lack of comparative values.

The surgeon must decide whether it is better to perform tonsillectomies in the summer months, when the incidence of infections of the respiratory tract is lowest, or to avoid the period of epidemics of poliomyelitis and run the risk of increased infections of the respiratory tract. Since most tonsillectomies are necessary surgical procedures in the interest of preventive medicine, the operation should not be postponed because of an unfounded fear of poliomyelitis. To aid in answering this question, Cunning urges that a study be made on children who are to be tonsillectomized this summer to determine whether they are more likely to contract poliomyelitis than nontonsillectomized children.

RYAN, Philadelphia.

Treatment, Neurosurgery

SUBARACHNOID ALCOHOL BLOCK IN PARAPLEGICS. C. HUNTER SHELDEN and ERNEST BORS, J. Neurosurg. **4**:385, 1948.

Shelden and Bors report the results of subarachnoid injection of alcohol in 24 paraplegic patients. All experienced immediate cessation of reflex spasms; in only 1 patient did the spasms recur, and then to only a minimal degree and limited to one leg. One had no return of spasticity for as long as thirteen months.

Eight of the paraplegic patients had hypertonic and 16 had atonic bladders. Of the latter, all but 1 was relieved to some extent of the complete retention. The hypertonic group responded even more favorably; all 8 patients acquired voluntary micturition, with an increase in capacity of the bladder to 300 or 400 cc.

Bowel disturbances, notably constipation or involuntary movement, followed alcohol block but were easily corrected by appropriate measures. The effect on sexual function was rather pronounced; 22 of the 24 patients lost the power of erection after injection. Of the 17 patients who had sexual dreams before the block, 5 lost the dreams completely and 1 had incomplete dreams.

The longest period of observation after intrathecal instillation of alcohol was fifteen months. During this time there was complete relief of the spasticity and

mass reflexes, and the beneficial effects on the bladder have been maintained in all the patients thus treated.

TOZER, Philadelphia.

Encephalography, Ventriculography, Roentgenography

PLATYBASIA. CLARA MAYERSZKY, J. Neurol., Neurosurg. & Psychiat. **12**:61 (Feb.) 1949.

Platybasia may be classified into primary basilar impression, which results from a defect in craniovertebral development, and secondary basilar impression, which results from diseases causing softening of bone, such as osteitis deformans, rickets and osteomalacia. The diagnosis can be made by roentgenogram, which in the lateral view shows an increase in the basal angle. Neurologic signs include paraplegia, quadriplegia, ataxia, nystagmus, dysphagia, dyspnea and dysarthria. The treatment is surgical. The author reports a case of platybasia secondary to rickets in which the condition responded to operative intervention consisting of upper cervical laminectomy and suboccipital decompression.

N. MALAMUD, San Francisco.